

The SCALP *in HEALTH and DISEASE*

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WITH 312 ILLUSTRATIONS

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TO
MY WIFE

FOREWORD

Friends will sometimes ask, 'Aren't you afraid you will get too one sided if you confine your interests entirely to dermatology?' The answer is that cutaneous medicine (and surely every other field of medicine as well) offers such diversified stimuli and problems that 'custom cannot stale their infinite variety'

There are the economic problems and the public health ones, the administrative problems and the research ones, the problems of pedagogy and of training, the problems of genetics and those of environment, the psychological problems and the anatomical ones, the problems of statistics and mensuration and those of intuition and imagination, the problems of helping the individual sufferer and the problems of creating organizations and structures to help communities—and so on for a list well nigh as diversified and as all inclusive in its scope as the sum total of human interests and opportunities

What has just been said of dermatology applies with almost equal force to that important segment of the specialty known as the hair. The hair is expensive—think of the millions spent on bleaching, tinting and dyeing it, on curling, on waving or on straightening it out, on cutting it and on shaving it off, on destroying it or on trying to make it grow, on making it lie flat or on making it stand up, to say nothing of the switches, transformations, chignons, and other supplements to fill out its deficits

These are all concerns of the "healthy" hair and one must add to them the enormous amount of care and money spent upon the hair when it is sick—so that it is quite likely that no other human structure can compare with the hair as an object of universal interest and as a cause of universal spending. The cost of upkeep of a nation's hair may perhaps rival that of a nation's army.

I cannot say that I agree with every word in this book of Dr. Howard Behrman's. As in every book of this scope, there are quite a few statements which allow for honest differences of opinion. But I can say that, as far as I know, this text is the most modern and encyclopedic work on the microcosm of the human hair. Where else will one find such up to date dissertations on the anatomy, chemistry and physiology of the hair, its endocrinology, anthropology and embryology, the diagnosis and treatment of its diseases, and the use and abuse of measures to alter its appearance? The present text goes into all these and many more matters in clear and useful fashion. It should therefore prove invaluable to students and physicians, dermatologists and nondermatologists alike—and also to many others who are interested in the problems of the hair and scalp.

Now that his book is completed, Dr. Behrman will assist in the development of a special "Clinic for the Scalp and Hair," in one of our hospitals, so that the host

of sufferers who require relief from troubles of the scalp and hair, and the many more who need advice about their care, can come and benefit from the knowledge Dr Behrman has acquired in these particular fields

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PREFACE

This book approaches the problems of the hair and scalp from a fundamental anatomic and physiologic viewpoint. The initial chapter includes a study of every known function and activity of the component structure of the normal hair apparatus. Although voluminous references have been consulted and are available at the conclusion of the chapters, the material has been condensed and presented in a simple manner in order to avoid the distraction of names, references, and extraneous material veering off at a tangent. The subsequent chapters deal with all the known disorders and diseases of the scalp, including anomalies, alopecias, and infections, scalp involvement due to systemic disease, scalp involvement due to other skin diseases, malformations, new growths, and neurodermatoses. The subject matter includes discussions of the most recent advances concerning the endocrines and hair growth, scalp nutrition, and the daily hygiene of the hair and scalp. The various types of hair preparations in common use, such as shampoos, dyes, bleaches, hair and wave lotions, and their effects on the hair and scalp have also been thoroughly reviewed.

Several equally important factors contributed to my decision to write this book. First of all, the idea prevalent among laymen and indeed even among some members of the medical profession, that diseases of the scalp are of minor importance, undeserving of protracted study and creative experimentation, is at variance with the facts. Often these scalp diseases are the early symptoms of skin disease elsewhere and of deep, systemic, or psychoneurotic disorders. Further, the disagreeable involvement of an appendage as much in the public eye as the hair is in itself the cause of serious feelings of inferiority, resulting in the loss of emotional stability and even at times impairing the ability of the individual to discharge his obligations of employment. Certainly these unfortunate patients deserve to have proper care along the lines of the most recent medical advances. The dermatologist, with his special training in diseases of the hair and scalp, is well aware of advances in this field, although no single source of recent knowledge is available to him. Too often the general practitioner, unacquainted with the latest therapeutic measures, embarks on a course of trial and error which may aggravate the condition and obscure the original disease by the time the patient is finally referred to a dermatologist. Had that general practitioner been able to consult one volume, an authoritative handbook containing the most modern contributions to the study of the scalp, such an unfortunate situation might have been avoided.

Dermatologists are not the only persons who must be cognizant of the disorders besetting the hair and scalp. The astute barber, the beautician, and the entire cosmetic industry can ill afford to be ignorant of the basic hair and scalp problems of the people with whom they deal in their daily occupations. These individuals, if acquainted with the fundamental facts of the subject, would be in an admirable

position to recognize a condition requiring medical care and so keep their clientele from passing into the hands of the untrained and unscrupulous by referring them to the proper medical channels. By so doing they could render a valuable service by protecting the individual against the deceptive claims of many hair preparations on the market which even the Federal Food, Drug and Cosmetic Act cannot completely control. Clever advertising maneuvers circumvent the law and claim results which are scientifically and physiologically impossible.

I believe that those who suffer from disorders of the scalp and hair have long been subjected to needless expense and anguish because of the lack of information regarding the cause and treatment of these conditions. It is my earnest hope that this volume will prove of value by presenting in simple fashion the most recent knowledge pertaining to the scalp in health and in disease.

HOWARD T. BLIERMAN

New York N. Y.

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During the preparation of this volume I frequently consulted various texts, extracting and incorporating information required for a work of such all inclusive scope. I am deeply grateful to the authors and publishers of the following books which were ever at hand for consultation on my reference shelves. Rather than list them in the bibliographies appearing at the close of each chapter, I prefer to declare my indebtedness to these general texts in this appreciative foreword.

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It is impossible to prepare a technical work without the assistance and criticism of colleagues specializing in dermatologic and related fields, and the cooperation of scientific publications. It is both my duty and pleasure to name those who have rendered me an invaluable service by allowing me to reprint quotations from their books and articles and to enrich the text with numerous photographs which would have been impossible for one physician to collect in a lifetime of practice. I wish to proffer my heartfelt thanks for their unselfish aid.

The author wishes to express a special debt of gratitude to Dr Lee McCarthy and to Dr Richard L Sutton, Jr, without whose kind assistance in obtaining numerous photographs and clinical material from their dermatologic textbooks, the publication of this volume would have been greatly delayed.

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CONTENTS

CHAPTER I		PAGE
EMBRYOLOGY, ANATOMY, PHYSIOLOGY	- - - - -	21
Embryology	- - - - -	21
The Rat and Mouse	- - - - -	21
The Human Being	- - - - -	28
Anatomy	- - - - -	31
The Scalp	- - - - -	31
The Hair Follicle	- - - - -	34
The Hair Shaft	- - - - -	35
Sebaceous Glands	- - - - -	49
Sweat Glands	- - - - -	55
Circulation	- - - - -	57
Innervation	- - - - -	59
Musculature	- - - - -	60
Arrangement Patterns and Regional Distribution	- - - - -	61
Anthropology	- - - - -	64
Gross Hair Form	- - - - -	65
Inheritance	- - - - -	65
Distribution of Hair Forms	- - - - -	66
Color	- - - - -	67
Cuticular Scales and the Medulla	- - - - -	67
Cross Sections	- - - - -	68
Hair Index	- - - - -	70
Areas	- - - - -	70
Hair Weight	- - - - -	70
Facial Hair	- - - - -	71
Pilometric Measurements	- - - - -	71
Alopecia	- - - - -	72
Variation	- - - - -	72
Physiology	- - - - -	72
Pigmentation	- - - - -	72
Rheologic Properties	- - - - -	83
Percutaneous Absorption and Permeability	- - - - -	92
Hair Exchange and Hair Growth	- - - - -	94
Endocrines and Hair Growth	- - - - -	96
Nutrition	- - - - -	109

CHAPTER II

	PAGE
NORMAL SCALP, HAIR PREPARATIONS, DERMATITIS	122
Care of the Normal Scalp	122
Brushing	122
Combing	123
Cutting	124
Singeing	124
Shampooing and Washing	125
Hair Preparations	126
Shampoos	126
Hair Lotions	129
Setting Lotions and Lacquers	131
Wave Lotions	132
Hair Creams and Fixatives	141
Hair Straighteners	142
Hair Dyes	143
Dermatitis	148

CHAPTER III

ALOPECIA	162
Congenital Alopecia	163
Congenital Anomalies Associated With Alopecia	168
Congenital Ectodermal Defect	168
Congenital Defects of the Scalp	168
Chondrodystrophia Calcificans Congenita (With Pseudopelade)	169
Congenital Skull Depressions	169
Congenital Auricular Fistula	171
Pachyonychia Congenita	172
Werner's Syndrome	172
Male Pattern Alopecia (Common Baldness)	173
Cicatricial Alopecia Due to Physical Agents	184
Mechanical	184
Cold	188
Heat	188
Ultraviolet Light	189
Roentgen and Radium Rays	189
Atomic Bombs	192
Medicolegal Aspects of Hair Morphology	193
Pseudopelade	200
Folliculitis Decalvans	200

CHAPTER IV

	PAGE
THE SEBORRHEIC DIATHESES - - - - -	220
The Pityriases (Simplex and Steatoides) - - - - -	220
Seborrhea Oléosa - - - - -	226
Seborrheic Dermatitis - - - - -	232

CHAPTER V

INFECTIONS - - - - -	245
Bacterial Infections - - - - -	245
Impetigo Contagiosa - - - - -	245
Impetigo of Boeckhart (Superficial Pustular Perifolliculitis) - - - - -	245
Furunculosis and Carbunculosis - - - - -	249
Lupoid Sycosis - - - - -	253
Folliculitis Keloidalis (Dermatitis Papillaris Capillitis, Acne Keloid) - - - - -	256
Dissecting Cellulitis of the Scalp (Perifolliculitis Capitis Abscedens et Suffodiens) - - - - -	260
Anthrax (Malignant Pustule) - - - - -	265
Erysipelas - - - - -	266
Infectious Eczematoid Dermatitis - - - - -	266
Tuberculosis - - - - -	267
Leprosy - - - - -	270
Spirochetal Infections - - - - -	272
Syphilis - - - - -	272
Yaws (Frambesia) - - - - -	284
Virus Infections - - - - -	285
Disseminated Cutaneous Herpes Simplex (Kaposi's Varicelliform Eruption) - - - - -	285
Herpes Zoster and Simplex - - - - -	286
Varicella and Variola - - - - -	286
Parasitic Infections and Infestations - - - - -	287
Vegetable Parasites (Fungi) - - - - -	287
Microsporum and Trichophyton - - - - -	288
Favus - - - - -	332
Piedra - - - - -	341
Systemic Fungus Infections - - - - -	346
Animal Parasites - - - - -	346

CHAPTER VI

SCALP DISORDERS OF PSYCHOGENIC ORIGIN (PROVED OR PRESUMPTIVE) - - - - -	356
Alopecia Areata - - - - -	357
Circumscribed Neurodermatitis (Suboccipital Dermatitis, Lichen Chronicus Simplex) - - - - -	375

CHAPTER II

	PAGE
NORMAL SCALP HAIR PREPARATIONS DERMATITIS	122
Care of the Normal Scalp	122
Brushing	122
Combing	123
Cutting	124
Singeing	124
Shampooing and Washing	125
Hair Preparations	126
Shampoos	126
Hair Lotions	129
Setting Lotions and Lacquers	131
Wave Lotions	132
Hair Creams and Finishes	141
Hair Straighteners	142
Hair Dyes	143
Dermatitis	148

CHAPTER III

ALOPECIA	162
Congenital Alopecia	163
Congenital Anomalies Associated With Alopecia	168
Congenital Ectodermal Defect	168
Congenital Defects of the Scalp	168
Chondrodystrophia Calcificans Congenita	
With Pseudopelade	169
Congenital Skull Depressions	169
Congenital Auricular Fistula	171
Pachyonychia Congenita	172
Werner's Syndrome	172
Male Pattern Alopecia Common Baldness	173
Cicatricial Alopecia Due to Physical Agents	184
Mechanical	184
Cold	188
Heat	188
Ultraviolet Light	189
Roentgen and Radium Rays	189
Atomic Bombs	192
Medicolegal Aspects of Hair Morphology	193
Pseudopelade	200
Follicular Decalvans	203

CHAPTER VIII

SCALP INVOLVEMENT DUE TO SYSTEMIC DISEASES

	PAGE
Calcinosis - - - - -	457
Hand Schuller Christian Disease - - - - -	457
Lipoid Proteinosis - - - - -	457
Lupus Erythematosus (Discoid Type) - - - - -	458
Lupus Erythematosus Disseminatus - - - - -	467
Pernicious Anemia - - - - -	467
Porphyria - - - - -	467
Scleroderma (Circumscribed Type) - - - - -	467
Sickle Cell Anemia - - - - -	472
Sjogren's Syndrome - - - - -	473
Vogt Koyanagi Syndrome - - - - -	473
Xanthoma Tuberosum - - - - -	474

CHAPTER IX

NEW GROWTHS - - - - -	476
Introduction - - - - -	476
General Surgical Considerations - - - - -	476
Benign Epithelial New Growths - - - - -	478
Cornu Cutaneum (Cutaneous Horn) - - - - -	478
Epithelial Cysts - - - - -	479
Epithelial Nevus - - - - -	482
Keratosis - - - - -	483
Molluscum Contagiosum - - - - -	486
Multiple Benign Cystic Epithelioma - - - - -	488
Nevus Epitheliomatocylindromatosus - - - - -	489
Nevus Epitheliomatosus Sebaceous Capitis - - - - -	494
Nevus Syringadenomatosus Papilliferus - - - - -	494
Psammoma - - - - -	495
Syringoma (Syringocystadenoma) - - - - -	495
Verruca (Wart) - - - - -	496
Woolly Hair Nevus - - - - -	496
Benign Connective Tissue New Growths - - - - -	497
Angioma (Vascular Nevus) - - - - -	497
Blue Nevus (Jadassohn Tische) - - - - -	502
Cicatrix and Keloid (Scars) - - - - -	503
Fibromas - - - - -	505
Granuloma Pyogenicum - - - - -	507
Lipoma - - - - -	509
Myxoma - - - - -	510
Osteoma - - - - -	510
Malignant Epithelial New Growths - - - - -	510
Basal Cell Epithelioma - - - - -	511

	PAGE
Basal-Squamous Cell Epithelioma - - - - -	516
Dermal Appendage Carcinoma - - - - -	516
Metastatic Carcinoma - - - - -	521
Nevocarcinoma (Melanotic or Amelanotic) - - - - -	522
Squamous Cell Epithelioma - - - - -	527
Superficial Carcinoma (Superficial Epitheliomatosis) - - - - -	529
Xeroderma Pigmentosum - - - - -	529
Malignant Connective Tissue Neoplasms - - - - -	529
Sarcoma Cutis - - - - -	529
Lymphoblastoma - - - - -	531

APPENDIX

FORMULARY - - - - -	542
Shampoos - - - - -	542
Hair Lotions - - - - -	543
Mildly Keratolytic and Rubefacient - - - - -	543
Moderately Keratolytic and Rubefacient - - - - -	544
Alopecia Areata - - - - -	547
Strongly Keratolytic and Rubefacient - - - - -	547
Ointments - - - - -	548
Moderately Keratolytic and Rubefacient - - - - -	548
Chronic, Scalp Disorders of the Scalp - - - - -	548
Lotions - - - - -	548
Ointments - - - - -	549
Seborrhea Oleosa and Seborrheic Dermatitis - - - - -	549
Ointments - - - - -	549
Lotions - - - - -	550
Bactericidal and Antiseptic Preparations - - - - -	550
Fungicides - - - - -	551
Pruritus - - - - -	551
Ointments and Lotions - - - - -	551
Psoriasis of the Scalp - - - - -	552
Hair Dressings - - - - -	552
Nonoil Fixatives and Hair Creams - - - - -	553
Hair Oils - - - - -	553
Brilliantines - - - - -	554
Liquid - - - - -	554
Solid and Semisolid - - - - -	554

THE SCALP
IN
HEALTH AND DISEASE

THE SCALP IN HEALTH AND DISEASE

CHAPTER I

EMBRYOLOGY, ANATOMY, PHYSIOLOGY

1. EMBRYOLOGY

Existing knowledge of the embryology of hair growth and development has been presented for many years with repetitious uniformity by the standard text Concepts theories, and even observations based on limited study have been accepted as factual data by the average student. Fortunately, many new and scientific studies pertaining to the various embryologic aspects of hair growth have recently been published. Butcher* has made an admirable survey of these studies in addition to his own important and basic research, and the following material is based on his survey.

THE RAT AND MOUSE

The development of the pilary system in our most common laboratory animals, the rat and mouse, is similar to that which occurs in human beings. Since many extensive studies have been made on these animals, their embryologic hair development will be described first. Hair germs become apparent in 17 to 20 day fetal rats as a crowding of cells, with a slight convexity, in the deepest layers of the epidermis. These germs continue to form from the stratum germinativum from birth until the age of 10 days.

The next stage in their development consists of an increase in their convexity and the appearance of a slight condensation of connective tissue, the prospective papilla, deep to them. Further development consists of an elongation of the epithelial bud whose base is capped by the enlarged prospective papilla. In a slightly later stage of development, which occurs typically in the 2 day old rat, the base of the epithelial portion has enlarged and invaginated to form the bulb of the hair, and the enlarged connective tissue papilla protrudes into the bulb.

*The author is greatly indebted to Dr. Earl O. Butcher, Professor of Anatomy, College of Dentistry and the Graduate School of Arts and Sciences, New York University, for permission to use this material compiled from his studies and research on the development of the pilary system as published in the *Annals of the New York Academy of Sciences*, March, 1951.

Beginning near the bulb the core of the epithelial column separates from the peripheral cells which become the outer sheath of the hair and retain their continuity with the stratum germinativum. The core forms the inner sheath and the shaft of the hair. In 5 day old rats some of the epithelial downgrowths have few indications of much differentiation while in many of them may be seen a definite hair shaft with inner and outer root sheaths.

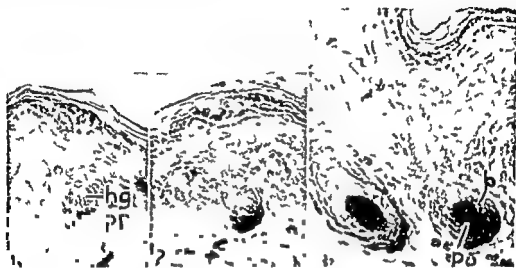


Fig 1—Section from 5-day-old rat showing larger and prospective papilla which contains phosphatase

Fig 2—Elongated bud and enlarged papilla with phosphatase

Fig 3—Section showing newly formed bulb and papilla containing phosphatase

Abbreviations: Figs 1-16: *b*—bulb *c*—core *fh*—first hair *fn*—folds in nuclei *hg*—hair germ *m*—medulla *n*—new cells *o*—dead cells *os*—outer sheath *p*—papilla *pp*—prospective papilla *sh*—second hair

(Figs 1-16 courtesy Dr Furl O. Butcher and Ann New York Acad Sci)

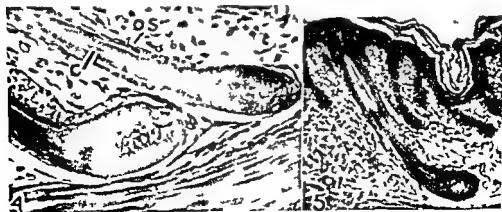


Fig 4—A fold in which the core is separating from the outer sheath cells

Fig 5—The continuation of the hair

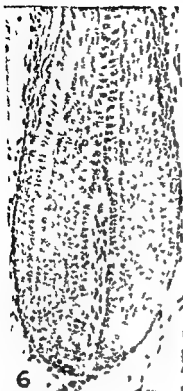


Fig 6—Section showing origin of the cells of the medulla from the matrix cells around the tip of the papilla

Fig 7—The pigment distribution in the hair of the black rat



Fig 8—Melanophores with dendroid processes extending into the cortical cells

As the hair and its sheath elongate, the central epithelial cells of the column distal to the cone move aside, allowing the tip of the hair to erupt. The inner root sheath, by its glassy appearance, contrasts sharply with the outer, especially near the apex where nuclei are absent or scarce. Huxley and Henle layers can soon be recognized in the inner sheath. The epithelial cells of the bulb resting on the surface of the papilla, represent the future matrix. This matrix, by active mitosis in that portion at the base of the papilla, gives rise to cells destined to become cuticle, cortex, and inner sheath, while the matrix cells around the tip of the papilla are pushed outward to become the medulla of the hair shaft.

The melanophores of black hair follicles are situated in the matrix, giving rise to the medulla. The melanophores are usually larger and contain more spherical nuclei than the other cells of the matrix. Dendroid processes of the melanophores, with their pigment, extend centrifugally into the forming cortical cells. Pigment is passed directly into the cytoplasm of the cortical and medullary cells by the pigment cell processes. Cells identical to the melanophores in black hairs are found in white hairs of albino rats and appear to be colorless pigment cells.

Two types of follicles may be found in the rat's coat. Some are very large and contain the longer and coarser hairs while the smaller follicles are more numerous and have shorter, finer hairs.

The matrix cells continue to add material to the elongating hair in the rat until the definitive length is reached at about the age of 17 days. In black hairs the formation of pigment stops a few days before the definitive length is reached and the diameter of the hair is gradually reduced.

This formation and elongation of the hair which ends about the 17th day of life constitutes the growing stage of the hair. At the age of 17 or 18 days the bulb of the hair begins a rapid transformation into a cornified, club shaped structure closely surrounded by a sheath. The hair club can be seen in the process of formation while the root is still quite long and a larger part of the inner root sheath still remains. The hair club slowly moves outward, stopping just below the level of the sebaceous gland. Desquamation of the inner root sheath occurs as it moves outward. The empty outer sheath forms an epithelial strand shortens and with the remnants of the papilla moves outward. The outer root sheath appears to pass into or is folded against the base of the hair club. At this time between the club sheath and papilla in the mouse, according to Dry, the hair germ of the next hair often becomes recognizable. This transformation into a quiescent condition occurs within an interval of 3 or 4 days and usually by the 21st day of life, the root of the hair is securely lodged in a resting follicle which remains inactive until the 31st or 32nd day of life. This inactivity extending from the 17th to the 32nd day has been designated as the resting period and with the growth stage constitutes a hair cycle.

The beginning of the growth stage of the next or second cycle on the 32nd day of life is marked by the appearance of a more definite hair germ at the base of the resting hair. Taylor in freezing experiments in the rat finds good evidence that most of the cells of the germ are proliferated from the base of the resting hair. He noted that the cells of the resting bulb had oval nuclei whose surfaces were thrown into folds. When these cells left the old bulb in the process of forming a new hair

germ, the folds in the nuclei persisted. Such folds enabled him to follow the origin of the cells in the new germ. Dry, as mentioned above, was able in many instances to recognize the hair germ much earlier in the mouse. The recognition of a hair germ depends upon the quantity of cells at the base of the resting hair. Apparently, in the mouse, as the resting stage is established, a considerable quantity of cells persists, while in the rat, only a few with growth potentialities remain closely folded at the base of the hair club. Simultaneously with the development of the hair germ, the prospective papilla appears, which is a condensation of dermal cells.

Among the cells that are contributed from the resting hair to the hair germ, a relatively small number may be distinguished from the rest by the development of long processes and a transparent cytoplasm containing minute melanin granules. These are the melanophores and, according to Taylor, they can be traced through consecutive stages to the fully differentiated pigment cells of the growing hair bulb. New pigment cells originate by the division of cells of the original melanophore stock received from the resting hair.

Some observers have taken the view that the same papilla can function in the growth of another hair. This may be true for vibrissae, for another hair is formed as soon as the growth of its predecessor is completed, and the original papilla appears to persist. Dry has been able to follow the papilla in the mouse by the accumulation of residual pigment in it. Just prior to the growth of the hair germ, the old papilla ruptures and a new one forms at the base of the follicle.

Growth is accomplished at first by the addition of more cells, from their respective sources, to those elements of the bud, and the bud then enlarges through mitosis, pushing deeper into the dermis. The advancing germ invaginates and envelopes the prospective papilla. Again, the matrix cells, or cells adjacent to the papilla, form a hair cone which separates from an external layer of cells, the external hair sheath.

The new hair usually does not push the old hair from the follicle but grows next to it. The fine hairs, or puppy hairs, produced in the first cycle, are thus joined by coarser hairs, which have the same quality, in subsequent cycles. This growing stage, as in the first cycle, lasts about 17 days and is followed by a resting period. Hair cycles in the rat, therefore, occur approximately every 34 days.

The cyclic growth occurs as a wave. Activity in follicles of the venter may be observed two to three days before it occurs in the dorsum. The wave begins in the venter, spreads dorsally, then anteriorly, and posteriorly. In the rat, it has been shown by skin transplantations that the follicles are receptive most of the time. Butcher poses the question: What is the controlling factor in the time of growth and the cause of the wave of growth?

Dry has observed a similar cyclic growth in the mouse. The first growth is completed at the end of a month, the second at two months, and the third sometimes at three months. The fourth generation is sometimes in progress on the venter before the third has finished on the dorsal region. According to him, one is unable always to see the hair germ of the next generation when inactivity, or resting, is being established. The papilla containing residual pigment disappears just prior to growth of the hair germ, and a new papilla is formed in which, with advancing age, residual pigment again accumulates.

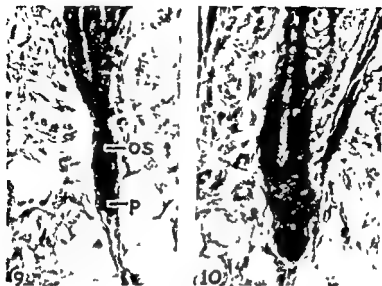


Fig 9 —The outer sheath and remnants of the papilla are moving outward

Fig 10 —The outer sheath and old papilla have approached the hair club



Fig 11 — The hair germ and prospective papilla of the second hair in a 3-day-old rat

Since most of the epithelial hair buds form from the stratum germinativum in the fetus or soon after birth the question arises if hair germs may form later in life from the germinativum or in case of the death of the follicle from any other source. In the rat most of the buds are formed prior to the 10th day of life yet

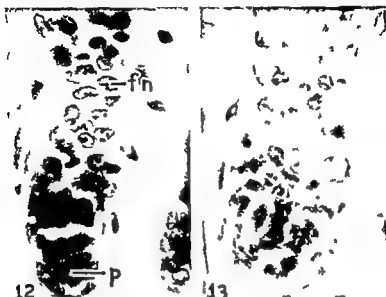


Fig 12—Folds or indentations in the nuclei of cells moving from the base of the old hair into the hair germ

Fig 13—A melanophore with long processes



Fig 14—A section showing the second hair pushing up beside the first hair

Fig 15—The cells of the old resting bulb were killed in freezing and appear as dense spots 'o'. New cells have migrated down from the epidermis and are the cells marked 'n'.

Fig 16—A new bud has formed from the epidermis independently of a pre-existing hair follicle in the skin that was frozen.

(Figs 1-16 courtesy Dr. Earl O. Butcher and Ann, New York Acad. Sci.)

areas denuded by injury often become repopulated. Taylor studied the margins of an area of skin of the rat following lethal freezing. The cells of the hair sheaths and those of the hair bulb had been killed by the freezing and new cells passed down from the old epidermis through the outer root sheath into the resting bulb for the new hair germ. The source of the melanophores was limited, however, to the old hair bulb and, once they were killed further pigmentation was lacking.

Taylor also observed the formation of new follicles from the stratum germinativum independently of an old follicle. It occurred however, only when the tissue underlying the new epithelium was old dermis or after newly formed dermis had become completely differentiated. These observations suggest that the differentiation of new hair germs may depend upon some inductive action of the underlying dermis. Hairless areas resulting from lethal freezing or from transplantation may thus be repopulated in the rat and the germinativum may possess hair potentialities throughout most of the life of the rat. Why should the epithelium lose deep growth potentialities while outward growth potentialities persist?

If the possibility of formation of new hair germs from the germinativum exists one may ask if there is any means of stimulating them. This problem is left for subsequent investigators who may present evidence that such stimulation may be irritants, hormones, etc. Butcher has presented evidence that the activity of hair germs at the base of resting follicles may be accelerated by adrenalectomy, by thyroxin administration or by the application of irritants. Confirmation of the effects of adrenalectomy has been made by Rall and Graef and Whitaker and Baker have inhibited activity of the hair germ by the percutaneous application of certain adrenal cortical preparations. In the discussion of the effects of endocrines on hair growth (pages 96-109) it is evident that estrogens, androgens and various adrenocorticotrophic factors also exert a specific influence. In the case of irritation as a method of accelerating the activity of the hair germs one must be extremely careful because the irritation causing acceleration may cause an acanthosis and parakeratosis in the epidermis.

Alkaline phosphatase has been studied in the developing hair. The enzyme is present to a very slight degree in the early epithelial bud becoming very abundant in the mesodermal papilla as the follicle elaborates the hair. The quiescent period of the hair cycle is characterized by a negligible amount of phosphatase. Glycogen in contrast to phosphatase is mainly distributed in the epidermal portion of the developing hair.

THE HUMAN BEING

Butcher states that growth and replacement of the hairs in the human being is similar to the process in the rat. About the only point at issue is whether the hair papilla persists or a new one forms. This point was discussed at great length by the older investigators. Some were inclined to believe that a new papilla formed and others believed that the new hair develops with the old papilla. Some believed that either might occur. Since the new papilla occupies exactly the same position as the old one it is difficult to understand why so many writers regard the formation of a totally new structure as essential to the process.



Fig 17—Progressive steps in the development of fetal hair. The hair germ stage is illustrated by *a*. The hair peg stage begins when the ectodermal downgrowth assumes a more or less cylindrical form as in *b*. The third stage (*c*) begins when the lower end of the hair peg is indented by the forerunners of the papilla in the corium. The fourth or sheathed hair stage (*d*) begins with the definite appearance of the hair shaft and lasts until the tip appears above the skin surface.

(Courtesy McCarthy, *Diseases of the Hair*, The C V Mosby Co, from Lewis's translation of Stohr's *Histology*, P. Blakiston's Son & Co.)

The first epidermal downgrowths of hair rudiments are found between the second and third months of intrauterine life on the upper lip, chin, and supraorbital regions. Between the fourth and fifth months, the hair begins to form in the hair rudiments of the face, and a month later the process of cornification has begun. By the sixth month, a lateral growth of the hair rudiment has evolved into a sebaceous gland, and another lateral outgrowth has formed to give insertion to the fibers of the erector pili muscle.

In the fetus, all the hairs belong to the lanugo type of hair. The shedding of the lanugo hairs in utero begins in the face and head of the fetus between the seventh and eighth months, and these hairs are replaced by new hairs which grow out of the old follicles (see Chapter III, *Male Pattern Alopecia*, page 173). These new hairs in certain regions such as the scalp, are long and stiff, while in the rest of the body new lanugo hairs take their place, forming the downy hairs of the skin. If this change has not taken place over the whole skin before birth, it is completed during the first few months of the child's life. At birth or soon after, hairs usually grow actively on the scalp. At puberty, hairs appear in certain regions, such as the beard, axillae and about the genitalia. In middle life and later, hairs, or vibrissae, grow about the nostrils and ears. In females after the menopause there is often an increase of pigmented and coarse hairs on the face, especially about the lips.

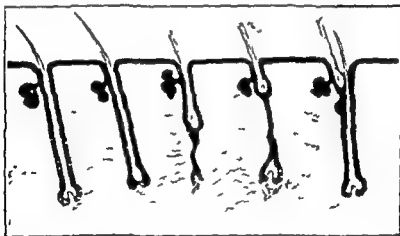


Fig. 18 Progressive stages in the maturation and replacement of fetal hair. (Courtesy McCarthy, *Diseases of the Hair*, The C. V. Mosby Co. after Danforth, *Hair*, A. M. A. Press.)

During life the hairs have a limited existence but they are not shed periodically over the entire body like the hairs of other animals. The hairs of different parts of the body have cycles of varying lengths.

In the scalp the hairs have been said to have an existence of from two to four years when they are shed and new ones are formed. How long the hairs generally grow and rest has not been recorded. Myers and Hamilton found that the average number of days required for the regeneration of hairs from the crown of the scalp was 129. In the region of the scalp above the ears it took 117 days for regrowth of hair. They also observed that the rate of growth of scalp hairs was 0.35 mm. per

day Of interest also is their observation that although scalp hairs grow faster in females than in males regeneration of hairs occurs with greater rapidity in males

Puppy hairs and, in the case of the human being, lanugo hairs, are produced first by follicles Then the follicles produce larger, stiffer hairs The follicle is thus able to produce hairs of different quality, but the means is unknown Fine hairs of lanugo quality are often present in baldness Many of them in all probability, occupy follicles where coarse hairs once grew What occurred to reverse the situation, or whether the follicle can be stimulated to form a coarse hair again is of great interest to all of us

Equally interesting are what controls the proliferative activity of the cells at the base of the resting club hair and to what extent these cells retain their growth potentialities By underfeeding experiments in the rat, it was shown that the proliferative activity remained after long periods of quiescence In hirsutism, the follicle retains its ability to produce a hair of a different quality after many years Is failure of replacement in the human being due to the loss of growth potentialities in the hair germ, to inhibition, to lack of stimulation, or to the lack of nutrition, or is there an inductive action in the connective tissue which is lost? These provocative questions as advanced by Butcher, constantly arise to plague us in subsequent chapters, and the incomplete answers to some of them may be found among the relevant material Although many interesting phenomena have been collected and analyzed, certain basic facts still prove elusive

THE SCALP

2 ANATOMY

The skin of the upper part of the head in association with its subcutaneous structure is known as the scalp It is limited to that integument which covers the top and back of the head and the two sides above and behind the ears Its detailed structure parallels that of the skin on other parts of the body except that it is securely fixed to the epicranial muscle by fibrous processes which pass through a middle layer of subcutaneous fat

The importance of the scalp is best seen from an examination of its five layers These are (1) skin (2) subcutaneous fat and fibrous tissue, (3) the epicranial (occipitofrontalis) and aponeurosis, (4) the subaponeurotic layer of connective tissue (5) the pericranium

The first three layers are connected to each other and move together The thick skin supported by the dense fibrous subcutaneous layer and epicranial aponeurosis is well adapted to protect the underlying cranium from the effects of trauma and in this connection the mobility of the first three layers on the subaponeurotic areolar tissue is important A scalp wound does not gape widely unless it involves the epicranial aponeurosis in which case it involves the subjacent dangerous area of the scalp so called because pus in this layer may spread widely underneath the scalp and even produce meningeal infection by spreading through the diploic or emissary veins In the process of scalping (whether performed by the knife or by the hair being caught in a mechanical device) separation takes place at this subaponeurotic layer which is loose delicate and devoid of fat

The average area of the adult scalp approximates 120 square inches. It is the site of a profuse growth of hairs varying in thickness, length, straightness, and color, depending on the individual's racial characteristics and sex.

Chemically, the human scalp has an unmistakably acid reaction varying between pH 4.5 and 5.5, due to lactic acid secreted in the sweat and to volatile fatty acids formed from the lactic acid by microorganisms in the scalp.



FIG. 19.—A and B (cross sections of normal scalp cut at slightly different angles. (Courtesy Dr. Carl Lavmon and Arch. Dermat. & Syph.)

THE HAIR FOLLICLE

The hair follicle is formed by the invagination of the epidermis and corium, sometimes even extending into the subcutaneous tissue. The dermic sheath derived from the corium consists of a fibrous coating of external longitudinal and internal circular connective tissue fibers, the latter lined by a hyaline layer continuous with the basement membrane of the corium. The sections of the follicle which originate in the epidermis are called inner and outer root sheath. In the lower two-thirds of the follicle, the outer root sheath is formed by the stratum germinativum and stratum mucosum, while the upper third depends for its derivation on the epidermal strata.



FIG. 21.—Section through lower third of hair follicle. *M* Medulla of hair. *CS* the cortical substance rich in pigment granules. *HC* the cuticle of the hair. *C* the cuticle of the follicle. *HL* Huxley's layer. *HF* Henle's layer. *OS* the outer sheath with external lower basal cells. *GL* the glassy layer of the hair bulb. *HP* the hair papilla with a vessel. *CT* connective tissue. (Courtesy of McCarthy. *Histopathology of Skin Diseases*. The C. V. Mosby Co.)

The inner root sheath is composed of three distinct layers. The innermost is a delicate cuticle containing a single layer of flattened imbricated cells with atro-

phied nuclei. Surrounding this cuticle is the middle layer known as Huxley's composed of a single or double layer of polyhedral nucleated cells. Finally the outer most, or Henle's layer, encloses Huxley's in a single stratum of nucleated cubical cells, both horny and fibrous in character. This entire inner root sheath disappears at the middle of the follicle, where the sebaceous gland secretion enters into the lumen of the follicle.

The outer, or dermic hair sheath is also composed of three clearly defined parts. Its innermost layer parallels the basal layer of the epidermis and is characterized as the hyaline or vitreous membrane. There is then a compact middle vascular layer, corresponding to the papillary layer of the skin. The final layer of the outermost sheath is constituted of elastic tissue loosely held in place with connective tissue. This corresponds to the reticular layer of the dermis.

The bottom of the hair follicle is molded on a vascular papilla originating in the corium and capped by the bulb of the hair which is a rounded extension of the hair root. The cells of the bulb are continuous with the outer root sheath and form the different parts of the hair itself as well as its inner root sheath.

In the hair follicle mitoses occur only in periods of activity, according to Leblond. In such periods mitoses become quite numerous throughout the lower portions of the Malpighian layers. These mitoses result in an outward motion along the hair axis of most components of the follicle. In the course of this motion the cells of the medulla and inner root sheath (soft keratin structures) desquamate. Thus only cortex and cuticle both of which are hard keratin structures, persist to form the emerging hair.

Montagna, Chase and Hamilton recently reported that nearly all of the components of the hair follicles are rich in glycogen. Beginning from the outside and proceeding inwardly, in the connective tissue sheath all of the fibroblasts contain glycogen granules. The external sheath throughout most of its thickness and length is virtually packed with glycogen. In the middle third of the follicle the tall columnar basal cells of the external sheath contain so much glycogen that apparently little cytoplasm is left. The basal cells become low cuboidal in the upper part of the follicle where the external sheath is continuous with the epidermis and in the basal part of the follicle around the hair bulb. In these regions all of the cells of the outer sheath become smaller. Concomitant with this decrease in the size of the cells there is a progressive loss of glycogen. The internal sheath contains no glycogen throughout its length not even near the hair bulbs. The cuticle of the internal sheath is also devoid of glycogen. The cells of the cuticle of the cortex are rich in glycogen in the region just distal to the hair bulb and approximately for one third the length of the follicle or less. In the middle third and distally where these cells are cornified there is no glycogen. In the cortex of the hairs glycogen is found in the region immediately distal to the bulb and spottily throughout the lower one fourth or one third of the follicle.

THE HAIR SHAFT

While the root of the hair is imbedded in this hair follicle its free portion is known as the shaft which like the follicle itself has three distinct parts. The

medulla or innermost section is composed of two to four layers of large cuboidal or modified polygonal cells loosely held together and containing keratohyalin, grains of pigment, fat granules, and scattered air spaces. Often the medulla does not extend the entire length of the hair shaft, appearing only intermittently. In the sections where it is absent, the hair consists of cuticle and cortex alone.

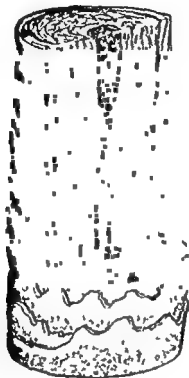


FIG. 22.—Semidiagrammatic cross section of the hair shaft. (Courtesy Reed, Den Beste, and Hummoller J. Soc. Cosm. Chem.)

The cortex or middle section is composed of longitudinally arranged fibers consisting of elongated closely applied fusiform cells containing pigment and air spaces. The cornification of the cortex which takes up the largest percentage of the hair structure is usually complete. When pigment granules are absent the cortex has a translucent appearance.

A layer of imbricated scales five fitting into the length of one form the outer part of the hair shaft known as the cuticle which begins at the upper portion of the neck of the papilla. The free ends of these flat cells are directed upward and interlock with the downward pointing cells of the cuticle of the follicle. The saw tooth action of the projecting free edges of the cuticle cells causes the shaft to travel in the direction of its root when the hair is rubbed between the fingers.

The hair chemically speaking owes its shape to the distribution of cystine which has been estimated to constitute approximately 15 per cent of the hair pro-

tein. Two amino acid groups joined together by two atoms of sulfur compose the cystine molecule. These cross linkages persist in an endless chain throughout the structural network of the hair and give it both form and specific characteristics.

In a study of 120 samples of human hair, the cystine, cysteine, sulfur, and nitrogen content was determined. It was found that there were more cystine and cysteine in male hair than in female, and that dark hair contained more cystine than light. Statistically, no significant variations in the nitrogen or sulfur content were observed.

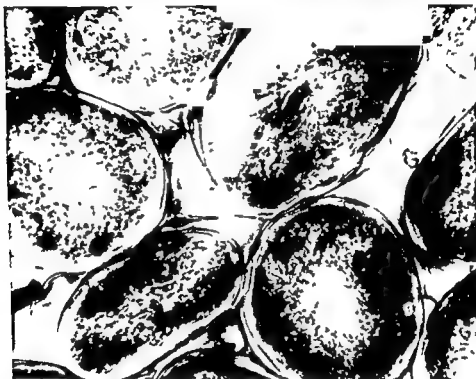


Fig. 23—Cross section of a group of hair fibers showing their irregular shapes. The cross section of the medulla can be seen in the fiber at the upper right. Note, however, that the medulla is completely absent in most of the fibers. (Courtesy, Mr Ray Reed)

Bollger tabulated the approximate amounts of uric acid, pentose, glycogen and total phenols in the aqueous extract of clipped mammalian hair.

	URIC ACID		PENTOSE		GLYCOGEN		TOTAL PHENOLS
	MG	%	MG	%	MG	%	MG %
Man	7	12	30		50		28

He stated that in addition to the insoluble keratin, the native extradermal hair fiber contains a number of water soluble organic compounds in physiologically large amounts. These include uric acid, glycogen, pentose, phenols, amino acids, urea, and ammonia. These compounds are considered to be by products of the process of

hair formation and there is some evidence that uric acid and pentose are derived mainly from the nucleic acid of degenerated nuclei.

In an analysis of the amino acids in hair keratin Block found cystine to be the outstanding component, with 16.0 Gm per 16 Gm of nitrogen. Other amino acids present in human hair include glutamic acid 14.8 (per 16 Gm of nitrogen), arginine 9.6, aspartic acid 8.0, serine 7.6, threonine 7.2, tyrosine 3.1, phenylalanine 2.7, lysine 2.6 and smaller amounts of methionine, histidine and tryptophan.

According to Grouard and Leblond the sulfur of the epidermis and its derivatives is mostly present as cystine residues in the Malpighian layer and cystine residues in the cornified layer. At the limit between these two zones the sulphydryl group of cysteine is oxidized to the disulfide bond of cystine, a process by which the keratin precursor present in the tonofibrils of the Malpighian layer is transformed into the completed keratin present in the tonofibrils of the cornified layers. The oxidation of cysteine into cystine probably provides cross links between the individual polypeptide chains making up the tonofibrils, thus welding them into a closely knit mass. This is at least one of the mechanisms by which the strength and chemical inertness of keratin may be explained.

This process is most elaborate in the formation of hard keratin derivatives such as the hair in which there is a pronounced enrichment of sulfur at the limit between Malpighian and cornified layers. This distinction as well as the difference in lipid content accounts for the physical and histologic peculiarities of hard and soft keratin.

At the present time we can distinguish between two types of keratin, namely soft and hard on a basis of their histologic appearance and chemical composition. According to Unna the cells of the stratum corneum have a thick hard wall and a fluid core rich in fats. It is probable that this fluid cell center renders the epidermal keratin somewhat supple and malleable, thus the name 'soft keratin'. Leblond states that the process of soft keratinization is indicated histologically by the presence of the granular and glassy layers, chemically by a low or only moderate amount of sulfur and physiologically by instability as evidenced by a spontaneous continued desquamation from the surface.

Hard keratin on the other hand is very tough and firm, has a high sulfur content, is physiologically stable and does not desquamate from the surface.

The Malpighian cells of the hair follicle are capable of producing 1) soft keratin structures, namely the medulla of the hair and the inner root sheath, both of which desquamate before the hair reaches the surface of the epidermis and 2) hard keratin structures, namely the cortex and cuticle, which emerge from the epidermis to form the hair itself.

According to Leblond Malpighian layers present in epidermal appendages such as hair take the form of stratified squamous epithelium, the basal cells of which are separated from the underlying connective tissue by a thin layer of fairly distinct reticular fibers. In areas of low biologic activity, such as the outer root sheath of the hair, the reticular fibers fuse to form a continuous basement membrane, whereas in areas of high activity, such as the Malpighian layer at the base of the hair follicle, reticular fibers are scarce or even absent. These facts suggest a relationship between the thinness of the reticular layer and the intensity of the meta-

bolic exchanges across such a layer from the subjacent connective tissue. The activity of the Malpighian layer is related to that of the subjacent tissue in many other ways. Thus it can be shown that the connective tissue beneath actively growing Malpighian layers, e.g., the hair papilla, is highly vascularized, contains many nuclei, and is rich in phosphatase and metachromatic substances, in contrast to the corium in general.

Leblond also observes that several components of the hair root produce soft keratin. Thus, the external root sheath, which is continuous with the epidermis, has granular, glassy, and cornified layers at least in the upper part of the hair root (i.e., near the opening of the sebaceous glands), while the lower part of this same external root sheath appears to be composed of dormant cells, which are producing little or no soft keratin. The base of the hair root consists of Malpighian cells, which form structures which grow parallel to the hair, namely, the internal root sheath on the outside and the medulla of the hair in the center. Both of these structures have a more or less distinct granular layer as well as areas of desquamation, and therefore must be producing soft keratin. Thus, the empty central canal often present in a fully grown hair can be explained as the result of desquamation of the medullary cells.

However, most of the Malpighian cells of the hair root produce the hair cortex, or hair proper, as well as the thin covering cuticle. Both of these structures are composed of hard keratin. Between the Malpighian zone and the fully formed hair, there is the gradual transition or keratogenous zone, characteristic of structures producing hard keratin.

Deviations From the Norm in the Hair Shaft—There are various deviations from the normal structures of the hair shaft which are not due to disease but to abnormal physiologic growth or mechanical peculiarities. One of these has been termed 'bayonet hair'. On a normal scalp 100 to 200 such hairs may be found in the region of the whorl. Where the scalp is beginning to develop an area of alopecia, great numbers of these hairs may be located at the margins of the bald spots.

A bayonet hair differs from the usual structure because of a spindle shaped swelling, 2 to 3 mm long, near its tip which gradually tapers off into the normal hair thickness. At times, two of these spindles may appear, one directly after the other on a single, otherwise normal, hair shaft. The thin end of the hair has a bend in it like a double joint, which gives the appearance of a bayonet protruding from the end of a gun.

Microscopic examination reveals squares or facets of pigmentation granules within each spindle which distort the usual arrangement of hair fibers. The cause of this structural deformity is unknown. It is believed that the point of the hair assumes a spiral form or is wound into a tight ball because the hair has taken more than normal time to emerge from its follicle and reach the skin's surface and so has become damaged by the internal pressure being exerted upon it.

Another structural deviation from normal hair growth is known as *monilethrix* or beaded hair. In this condition there is a marked keratosis at the mouth of the hair follicle which imparts the feeling of a grater to the examining finger. Irregular hairs like short bristles from 2 to 6 mm in length cover the scalp and slant in all directions. Each hair shaft is moniliform (i.e. resembling a string of beads),

and where the hair manages to attain any length at all, there are about six spindle-shaped thickenings along the single shaft

Histologic examination shows that intermittent nodes, darker in pigment than the slender, intervening portions consisting of a cortex and a thickened cuticle, contain the medulla, which is otherwise absent. They are often spirally twisted. Hairs of this nature are separated from the scalp with the slightest traction since they seem to have lost their usual attachment to the papilla and are merely held in place by the keratogenous mouth of the follicle, which has been sufficiently blocked to keep the hair from falling out without being drawn. The nodules may also be seen



Fig. 24.—Monilethrix in boy aged 11 years. The extreme degree of alopecia and follicular hyperkeratosis are characteristic. (Courtesy McCarthy, *Diseases of the Hair*, The C. V. Mosby Co. Original photograph by Dr. N. Tobias.)

within the follicular section of the hair, proving that the structural change begins early in the hair's development and is in no sense the result of an alteration of the free hair shaft above. Hairs of this beaded type never attain maximum growth since they break off at the atrophied internodal sections, either cleanly or with a brushlike fracture.

Monilethrix seems to be an hereditary manifestation, sometimes traceable through many generations of the same family. Its precise cause is unknown although many theories have been advanced. These include possibilities such as an hereditary lesion of the nervous system, concomitant constrictions of the cutaneous

muscles alternating periods of dilation and contraction of the papillae and an inherited disturbance of the nutrition of the follicle caused by trophic or vasomotor influences in the papillae. Actually we know little more than that it is probably the result of an unknown deforming pathologic change within the hair follicle. Therapy is of little value in this disease. Various measures have been recommended such as the local application of tar, sulfur and similar chemicals. Favorable results following x-ray epilation have also been reported. I have seen no dramatic change following any therapeutic regime including x-ray epilation.



Fig. 25.—Typical instance of monilethrix. (Courtesy Dr. Marion Sulzberger.)

Trichonodosis or the spontaneous knotting of the hair is another variant of normal hair structure which gives the hair shaft a nodular appearance although actually these nodules are true knots or loops usually situated in the peripheral half of the hair shaft. Splitting and breaking occur because of the strain exerted on the tensile strength by the tightness of the knot so that hair with this characteristic remains short. Examination of the fractured ends reveals that the shaft has either been split into several fibers resembling a brush or are pointed and atrophic or frequently bent into the form of a fishhook. Most of them have curled ends looping one or more times.

Cross sections of these hairs show them to be flat or oval forms associated with dry, curly hair, and some are twisted on the long axis. The two most usual types are the true knot and the slip knot. The latter may be promptly eliminated by pulling gently on the hair in the same way as one would remove a slip knot in a length of thread. No one has adequately explained the etiology of this condition beyond advancing the supposition that very dry curly hair frequently brushed and combed, or even exposed to the normal friction of lying down, may twist and knot itself from causes which are entirely mechanical. Curly hair made still curlier by brushing may form the loops which lay the basis for knotting at a subsequent time.

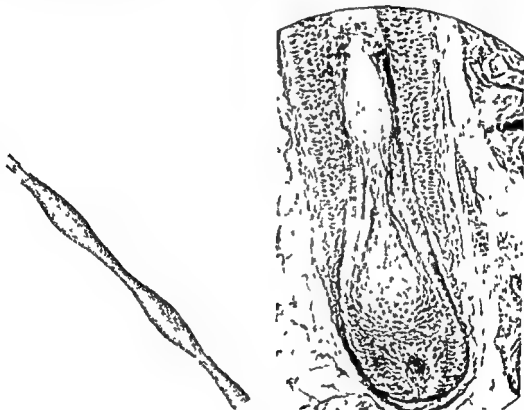


Fig. 26

Fig. 27

Fig. 26 Monilethrix. The hair shows the typical beading characteristic of the disease.

Fig. 27 Monilethrix showing typical changes in the hair follicle. (Courtesy Sutton and Sutton, *Diseases of the Skin*. The C. V. Mosby Co. Original photograph by Drs. G. M. Mackee and I. Rosen.)

The name *trichoclasis* was formerly used to include all the different types of transverse and longitudinal splitting and breaking of the hair shaft. At present we confine ourselves to the terms *trichoclasis* (transverse fracture), *trichoptilosis* (longitudinal fracture) and *trichorrhexis nodosa* (nodular swellings often with transverse fracture).

While the older literature states that *trichorrhexis nodosa* may be an aftermath of disease more recent findings indicate that this is infrequently true. The condition is due to an unknown process (probably traumatic) which changes the normal hair structure, and therefore it can be classified with other deviations. Like monilethrix the hair shaft has intermittent nodules but unlike monilethrix these nodules are gray and white. Brushlike fractures take place within these swellings, splitting in such a way that the broken fibers resemble the interlocking whisks of two broom heads. The underlying scalp is entirely normal.

This condition is probably caused by excessively dry and brittle hair subjected to overly frequent brushing, washing with soaps of a high alkaline content, lotions containing thioglycolates and ammonia (i.e., waving lotions), dyes and bleaches which include peroxide, and other chemical solutions employed on the scalp.

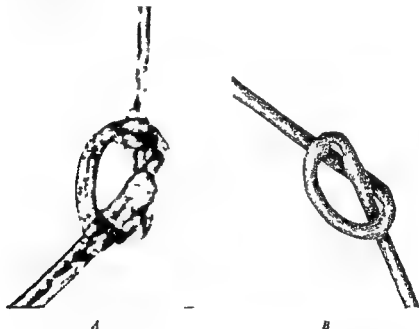


Fig 28 —Trichonodosis or knotting of the hair. A Typical hair. B drawing of usual type of knot. (A Courtesy Dr. Frank C. Combes and Dr. M. Scott.)

Trichoptilosis means a longitudinal swelling and splitting of the hair shaft but without the nodules noted above. Frequently the whole shaft is involved and looks like a fringed stem. The cause seems to be friction applied to excessively dry hair as well as all the corroding elements mentioned above. Local disease processes affecting the scalp and hair may also produce transverse or longitudinal fractures of the hair shaft.

Twisted hairs (pili torti) is the name given to a group of rare pilar anomalies characterized by twisting of the hairs on their axis. It may be accompanied by dryness and brittleness of the hair shaft with a moderate degree of alopecia. The relative baldness and the frizzy haired appearance are peculiar, and the linear twisting

which in many hairs is ropelike is readily observable and easily differentiated from that which occurs in monilethrix. Microscopically there is a slight twisting of the hair bulb with some degree of atrophy of adjoining sebaceous glands and follicles. The pathogenesis of this congenital anomaly is obscure. The dryness and fragility of the affected hairs is probably due to the impaired secretion of sebum. Therapy is of no value although a short haircut minimizes the cosmetic defect.

Trichostasis spinulosa, as first described by Galewsky, is a peculiar disorder affecting the hair follicles of the shoulders and back and the nape of the neck, near the scalp margins. The follicles contain blackish elevated, horny, spinous plugs, which fill the dilated orifices but can easily be removed. These keratotic masses contain bundles of lanugo hair which protrude beyond the keratotic plugs.



FIG. 29

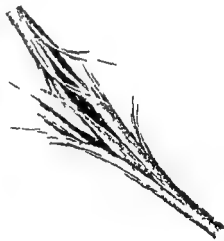


FIG. 30

FIG. 29 Alopecia of the posterior half of the scalp in which the hairs all show trichorrhexis nodosa and irregular contour of their shafts. Occasional hairs show a certain degree of atrophy of their bulbs. (Courtesy McCarthy Diseases of the Hair. Original case of Dr. Sandler.)

FIG. 30 Trichorrhexis nodosa showing transverse fracture of the hair shaft as well as fraying of the ends and longitudinal splitting of the shaft.

Hochstetler believed that either one of two sets of conditions might explain the abnormality. (1) there is an abnormal congenital upposition of the papilla of the follicle such as normally occurs in the pig or else there are multiple papillae or (2) all of the hairs are produced by one papilla but the hairs are not expressed from the papilla as they are formed but remain in situ and form the brushlike bundle.

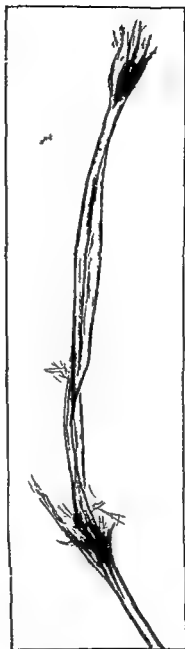


Fig 31—Trichorrhexis nodosa and trichoptilosis in the same hair (Courtesy McCarthy Diseases of the Hair)



Fig 32 — Pili torti in a 7 year-old girl (Courtesy McCarthy, *Diseases of the Hair* Original photograph by Dr F Ronchese)

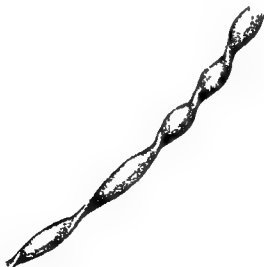


Fig 33 — Twisted hair (pili torti), showing characteristic features

Burgess believed that the condition represents a development late in life of a congenital malformation of the follicles

The deformity is a congenital one and treatment is only palliative

Acquired progressive kinking of the scalp hair is the name given to an unusual anomaly of hair growth and hair color reported by Wise and Sulzberger in 1932. The patient was a man aged 21 years, whose light brown, straight, and exceptionally vigorous head of hair had begun to undergo a change with the result that the frontal, temporal, and parietal portions of his scalp hair had become "woolly and kinky," and had changed in color from a light brown to black. A thorough examination of this patient disclosed no physical findings to explain this change, although he had



Fig 34—Twisted hair and monilethrix. 1 and 2, Twisted hairs, 3, moniliform. (Courtesy Sutton and Sutton, *Diseases of the Skin*. Original photograph by Dr F Ronchese.)

an enlarged thymus and was of the suprarenal type with an associated enlargement of the thyroid gland. The changed hair was thick, black, and kinky and of a woolly, almost matted appearance. Microscopically, the kinky hair shafts were much thinner than those of the straight hairs and a small proportion of them presented little globular and roughly spindle shaped swellings of the shafts, as well as trichorrhexis. Histologic studies of the scalp showed no alterations that might be construed as explaining the clinical change.

Wise and Sulzberger were impressed with the resemblance of this "acquired" form of kinking of the scalp hair to the congenital examples of the disorder previ-

ously reported by Wise as woolly hair nevus (page 496). However, as they observed, one of these congenital cases showed an associated linear verrucous nevus of the neck and arm which actually fused with a patch of kinky hair at the back of the neck, leading to the supposition that in this instance they were actually dealing with a compound nevus. They cite Meirowsky's explanation of the systematization of birthmarks, as follows: "The systematization of birthmarks is determined by the germ plasma. It is an ancient, phylogenetically observed racial peculiarity of



B

FIG. 35. A and B. *Trichostasis spinulosa* showing two different types of arrangement in horny case. A (Courtesy Sutton and Sutton Diseases of the Skin. Original photograph by Dr. J. H. Mitchell). B (Courtesy McCarthy Diseases of the Hair. Original photograph by Dr. Nobl).

man. These aberrations from the normal skin structure occur in the systematization, for example, loss of pigment—hair, gland or other changes—that systematization which is present in all normal skins becomes apparent. In general, this systematization has the characteristics of the markings found in animals.²²

This explanation suggested the possibility of a nevus tarda to the authors. They felt that the similarity of the findings in this case with those of woolly hair nevus seemed to favor this point of view. Moreover, the localization of the hair changes would favor an anomaly due to changes in the germ plasma as transmitted in the form of mosaic inheritance ("blazing" in animals) as suggested by Weidman in a previous discussion of Wise's second case. Against this concept were the facts that the condition was slowly progressive, that it was accompanied by some evidences of endocrine change, and that no nevus characteristics were present in the microscopic sections. Unfortunately, the rarity of this disturbance and the lack of subsequent reports have shed no further light on the subject. A study of various anthropologic reports, including the woolly hair mutations observed in animals and human beings, suggests the possibility of a delayed mutational variation as a cause of this rare disorder. In addition, it is not generally appreciated that some kinky hairs may occur here and there on the head and body of most Caucasians.



Fig. 36—Acquired progressive kinking of the hair. (Courtesy McCarthy, Diseases of the Hair. Original photograph by Drs. Fred Wise and Marion Sulzberger, Arch. Dermat. & Syph.)

SEBACEOUS GLANDS

At the end of the second month of intrauterine life, when the hair papilla protrudes obliquely into the epithelial cylinder, on the side of the obtuse angle thus formed two rounded projections develop. The upper outgrowth consists of an epidermal offshoot, an extension of the cells of the outer root sheath. By the fifth month its deep end becomes enlarged, lobulated, and undergoes a fatty transforma-

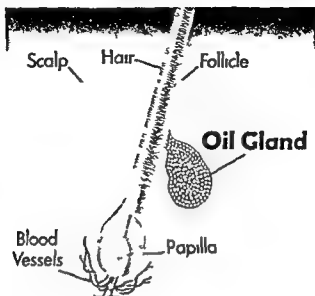


Fig. 37 —Schematic drawing of the pilosebaceous apparatus (Courtesy Mr. R. Reed and the Toni Company.)

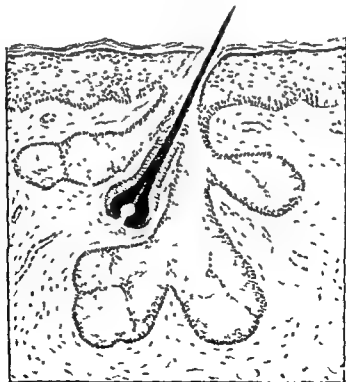


Fig. 38 —A lanugo hair of the glabrous skin with accompanying multilobular sebaceous gland. Note superficial location of hair follicle and the fact that the minute hair is really an appendage of the gland. (Courtesy McCarthy, Diseases of the Hair.)

tion thus developing into the secretory section of the gland. The narrow neck which connects this part with the follicle becomes its duct. During the last months of fetal life the sebaceous secretion and the cast off epidermal cells collect on the surface of the body and are called the smegma embryonum.

Most sebaceous glands are directly associated with hair follicles, since their ducts composed of cuboidal epithelium usually find outlets in the upper thirds of the follicles. Nevertheless there are some sebaceous glands elsewhere on the body surface which appear independently of the hair follicle.

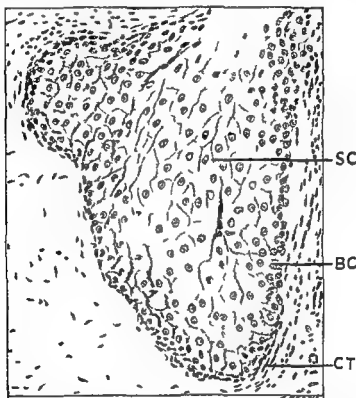


Fig. 39.—Sect on through a normal sebaceous gland. SC Sebaceous cells. BC basal cells. CT connective tissue cells as a pseudomembrane on which the basal cells rest. (Courtesy McCarthy: Histopathology of Skin Diseases.)

All through the corium existing in every part of the body except the palms, the soles, and the terminal phalanges we find these glands, each one of which is composed of a fibrous capsule, a membrana propria, and various epithelial elements. Their membranes are prolongations of the vitreous layer of the epidermis or correspond to the basal layer of the epidermis. Other cells conform to the types within a prickle cell layer of the epidermis or the external sheath of the hair follicle.

The size of the sebaceous glands is unrelated to the hair follicles to which they are usually linked. They vary from small simple saclike alveoli to larger multilobular grape-like constructions. Either a single or a double layer of columnar or

cubical epithelial cells form the periphery of the alveolus, whose center is occupied with larger, cuboidal polyhedral cells, containing varying amounts of fat. The smallest glands are found in the scalp, where there is maximum hair growth, and because of their numbers they have no room in which to grow. As the hairs decrease, the glands become larger and the hairs are permanently lost. The glands persist only until the final deterioration of the follicle.

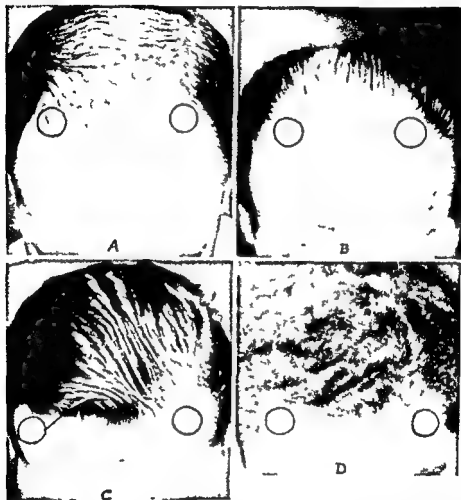


Fig. 40—A Sparse hair and much dandruff (age 42) B Little hair on the vertex and a very oily scalp (age 41) C Slight hair recession and small amount of dandruff (age 27) D Very good hair (age 25) (Courtesy Drs. F. C. Butcher and J. P. Parnell and J. Invest. Dermat.)

As fine granules of fat gradually become more numerous within the inner core of cells lining the gland, the nucleus of each cell disappears and the cells burst, releasing the fat or sebum which escapes into the duct of the gland. New cells are continuously supplied by the activity of the proliferating basal elements. The constant pressure of accumulating fat granules pushes the fat mixed with various

cellular debris through the duct up to the skin's surface. In the case of lanugo hairs and independent glands the sebum does not excrete through a hair follicle but directly on the surface.

It has been discovered by scientific measurements of the amounts of sebum discharged at the hairline of a group of individuals taken once weekly for eight weeks that a person with good hair and a small amount of dandruff averaged 0.160 mg of fat and 0.0119 mg of cholesterol per square centimeter the cholesterol amounting to 7 per cent of the total sebum. During these experiments it was made evident that frequent removal of the sebum did not increase the oiliness of the scalp.

An investigation of the physical properties of sebum proved that its viscosity increases as the sebum is cooled. An abrupt increase in viscosity was apparent as the temperature of the sebum was lowered to 29° to 30° C. a usual figure for the surface of the forehead. When the temperature was dropped to this level the

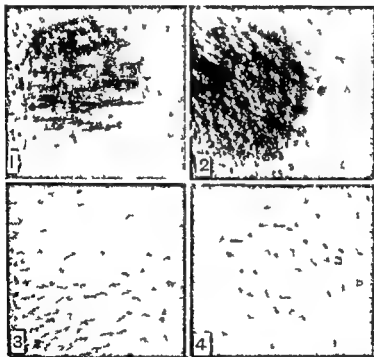


Fig. 41—1 and 2 Sebum which accumulated in an interval of twelve hours at the orifices of the sebaceous glands and in the sulci of the skin. It has been blackened with osmic acid vapors.

3 Skin which was cleaned with ether and immediately exposed to osmic acid vapors. No sebum appears to be present.

4 Sebum which accumulated in an interval of fifteen minutes. Sebum is present in the orifices of many of the sebaceous glands and has spread in the sulci in a few instances.

These illustrations show that the sebum on the forehead is not confined to the orifices of the sebaceous glands but spreads along the sulci between the orifices especially following an increase in temperature. (Courtesy Drs. E. O. Butcher and J. P. Parnell and J. Invest. Dermat.)

sebum collected in a capillary tube began to separate into its component parts, leaving a resinous like solid adherent to the side of the tube. Since the sebum cools as it reaches the surface of the skin, its increased viscosity provides a natural resistance to further exudation of sebum from the orifice of the gland, in this way controlling the level of the sebaceous coating. Warming the skin lowered the viscosity of the sebum, removing normal resistance and making it possible for more sebum to reach the skin's surface. The specific gravity of sebum collected from the foreheads of various persons was found to be 0.911.

There are various other factors which increase the amount of secretion: excessive intake of carbohydrates and fats in the diet, impairment of general health, and the developmental changes in the endocrine glands. Sebaceous secretion is stimulated at puberty as well as in the latter months of pregnancy, but as the individual grows older there is less and less secretion, until in old age we observe a characteristic dry skin and scalp.

Excessive amounts of sebum are excreted in the late stages of paralysis agitans (parkinsonism). The skin of the face becomes unusually greasy, converting it into a waxy mask. This change is due to a disturbance of the brain centers controlling secretion of sebum and definitely indicates the presence of a central control mechanism.

In an attempt to answer the question as to what are the fatty acids and acid yielding lipoids present in the skin's secretion it was discovered that those regions supplied by sebaceous glands contain both long chain and short chain fatty acids characteristic of sebum. Fat and fatty acids ranging in chain length from 7 to 22 carbon atoms predominate in the sebum's chemical composition. Olein, palmitin, stearin and their respective acids are all present accompanied by some inorganic salts, epithelial debris, cholesterol, a minute amount of ergosterol and water. Under certain conditions bromine and iodine up to 49.0 per cent have been located in analyses of sebaceous secretion.

Among the compounds which do not appear to have been hitherto identified as constituents of sebum are squalene and vitamin E. Squalene, an unsaturated hydrocarbon ($C_{30}H_{50}$) found in the liver oils of sharks and certain other fish and also in yeast was present to the extent of about 5 per cent of all the mixed samples of sebum studied by MacKenna. It is possible, of course, that excretion of squalene in the sebum merely represents the elimination of a metabolically useless compound but the possibility of squalene being an important metabolite should not be overlooked. Squalene has some structural chemical relationship with the carotenoids and also with some of the fat soluble vitamins (e.g. A and F) and the sterols and steroids. When squalene is administered to rats there is a very marked increase in the amount of unsaponifiable matter including cholesterol in the liver but the available evidence while not conclusive suggests that squalene is not converted into cholesterol.

It is of interest to note that Flesch has found that squalene and a group of lipid soluble unsaturated compounds with $C=C$ groups cause reversible complete baldness when applied to the skin of laboratory animals. These compounds include the synthetic dimers of chloroprene and certain esters of allyl alcohol, naturally occurring vitamin A, oleic and linoleic acids. He has brought up increasing

evidence that the depilation is caused by the reaction between the $-C=C-$ bonds and the sulphhydryl groups of epidermal proteins, probably an alkylation of the sulphhydryl group by the unsaturated double bonds in the molecule. Although he has not been able to depilate human beings with any of these unsaturated depilatory agents, these findings have suggested a working theory that human sebum has a profound influence on hair growth and keratinization. This theory is supported by the following indirect evidence:

- 1 A single application of sebum causes reversible hair loss in laboratory animals with profound changes in epidermal growth and keratinization.
- 2 Three of the depilatories (squalene, oleic and linoleic acids) are normal constituents of sebum.
- 3 Since chronic exposure to two unrelated depilatory compounds (the dimers and vitamin A) cause loss of hair, it is conceivable that one or several of the unsaturated components of sebum have a similar action when hair is subjected to their toxic effects over long periods of time.
- 4 It is known that male sex hormones are essential for the development of common male baldness, on the other hand, male sex hormones are the most powerful known stimulants of sebaceous secretion (see section on Endocrines and Hair Growth, page 99). Additional evidence for the role of sebum in the etiology of baldness is still in the preliminary experimental stage.

The secretion of significant amounts of vitamin E (tocopherols) in the sebum is also one of considerable interest, particularly in view of the recent attempts to treat certain skin conditions by the oral administration of this vitamin. Vitamin E is an antioxidant, and the presence of such a compound in the sebum should undoubtedly help to retard the oxidation of unsaturated fatty acids and related compounds in sebum, but how far this is of physiologic value cannot be decided at the present time. There appears to be a sufficient general evidence available, however, to suggest that vitamin E and other antioxidants of sebum may be of some importance in the maintenance of the normal condition of the skin.

The function of these secretory glands seems to be to supply a protective, oily coating which imparts a gloss to the hair at the same time that it guards against the dangers of exaggerated atmospheric changes. The effects of endocrine secretions on the amount and type of sebum produced is discussed in the section on endocrine physiology (pages 96-109).

SWFAT GLANDS

Early in the fifth month of fetal life, on the palms and soles the sweat glands grow downward perpendicularly from the stratum mucosum. They do not appear until much later over those portions of the body which produce hair. They extend through the corium and coil up in the subcutaneous tissue to form the secreting mechanism of the gland whose ducts do not open onto the skin's surface until the seventh month.

Sweat glands appear on all sections of the human body and are innumerable though it has been estimated that each individual has over two million of these

glands, which outnumber the hairs by five or six to one. There are approximately 2,700 to the square inch on the palms and soles, 1,300 in the areas in the region of the forehead, and 550 on the cheeks.

There are two varieties of sweat glands—the apocrine and the eccrine. The former are large coil glands appearing chiefly in the axillary, pubic, areolar, and anal regions at about the time of puberty. They are intimately associated with the hair follicle into which they empty not only fluid but a part of the cellular structure of their own inner layers. According to some investigators apocrine glands have also been found in the scalp. It has been suggested that *perifolliculitis capitis abscedens et suffodiens* (dissecting cellulitis of the scalp) is due to an infection in such aberrant glands. This observation may account for the occasional report of this condition in association with *hidradenitis suppurativa*, a deep seated infectious disease of apocrine glands elsewhere on the body surface.

The eccrine glands are small coil glands which cover the scalp, secreting only fluid and, unlike the apocrine glands leaving their cells intact after excretion. The body of these eccrine glands is composed of simple coiled tubes located in the dermis. These tubes are constructed of cubical glandular cells resting in a flattened spindle shaped layer of myoepithelial cells whose contraction helps secrete the fluid. The *membrana propria* surrounds this layer supported by a sheath of thick fibrous and elastic tissue. Blood vessels and connective tissue elements separate the glands from each other.

Each sweat gland possesses its own duct composed of two rows of cuboidal cells and an external connective tissue sheath. This duct leads upward from the coil gland proper to the subepidermis taking a spiral course through the corium. Since the funnel shaped outlet through the epidermis known as the sweat pore is not lined the fluid secreted is a combination of the product of the sweat gland to which has been added fluids from the adjoining intercellular spaces and even sebum. An acid reaction is normal with sodium chloride its main chemical constituent accounting for the salty taste. Among the other chemicals found in the fluid are urea and also a trace of sugar. Other inorganic components include creatinine, uric acid, ethereal sulfates of indole and skatole and albumin. When sweat is being secreted profusely it is apparent in liquid form otherwise it is given off in vapor and is not apparent. Between 500 and 1,000 cc. are separated from the body each day in either one of these two perspirations—visible and invisible.

The contraction of the myoepithelial cells may be partly responsible for the secretory discharge but since they probably have an ectodermal and not a mesodermal origin it is problematic whether or not they could be unstriated muscle fibers. The sympathetic nervous system activates the sweat glands since the cutaneous sensory nerves are closely connected to the sympathetic secretomotor fibers. Should this nerve supply become discontinued for any reason the sweat glands are no longer able to function. The secretion of one sweat gland since it is dependent on the nervous system is not continuous its discharge being intermittent and in the form of minute drops.

Hyperhidrosis Capitis. Excessive sweating of the scalp is not uncommon and often gives rise to unpleasant odors augmented by infrequent washing and perfumed mixtures. Proper local hygiene and shampooing of the scalp will usually

minimize this disorder. In the occasional instance of excessive malodorous sweating of the scalp, the author has successfully employed Banthine to cut down the activity of the sweat glands via control of the autonomic nervous system. The dosage is approximately 50 mg every six hours until control is established and then a progressive decrease until the determination of a satisfactory maintenance dose.

CIRCULATION

The hair follicles and the sweat and sebaceous glands must receive an abundant supply of blood for their nourishment. The main sources of this supply are vessels situated in the subcutaneous tissue. The internal carotid artery supplies those arteries servicing the frontal region, while the external carotid takes care of those vascularizing the rest of the scalp. These two sets of branching vessels connect freely with one another by means of collateral channels and also across the median plane.

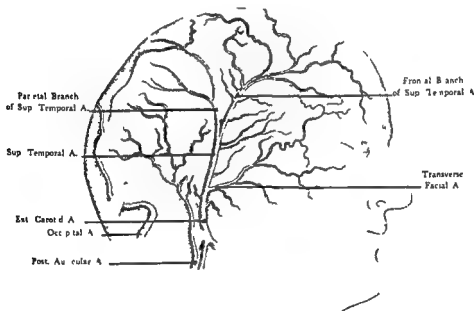


Fig. 49—Arterial supply of the scalp

Two parallel vascular plexuses are horizontally placed—a coarse one forming an arterial rete in the deep part of the corium from which twigs supply the fat lobules and sweat glands while the other finer and more delicate, spreads a network in the subpapillary layer from which it services the sebaceous glands. Both of these nourish the hair—the subcutaneous plexus providing for the hair papillae whose contracted necks act as points of entrance for the blood vessels and the

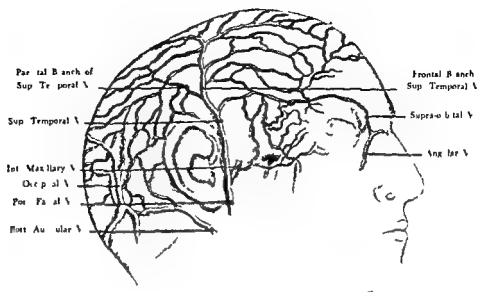


Fig. 43.—Venous drainage of the scalp

subpapillary plexus feeding the follicle by means of its arterial capillary loops in the surrounding connective tissue. The blood vessels do not possess a consistent pattern. Abnormally elongated sweat glands and unusually large hair follicles often have a double blood supply.

While the arteries and their various tributaries take care of the blood's distribution, five veins are responsible for its drainage from the superficial portions of the scalp. These are the frontal, the supra-orbital, the superficial temporal, the posterior auricular, and the occipital. Lymph vessels abounding in the adjacent connective tissue must supply some lymph to the hair's outer sheath, although no lymph vessels have been located which supply the hair directly through its follicle. The epidermis has no blood supply whatsoever in contrast to the cutis and subcutaneous tissue, which are characteristically highly vascular.

The lymphatics of the scalp form a rich network in the neighborhood of the vertex, from which vessels pass in various directions. From the frontal region a number of vessels pass downward and backward to the parotid nodes; those from the parietal and temporal regions pass to the anterior auricular, parotid, and posterior auricular nodes; and those from the occipital region pass partly to the occipital nodes and partly to the superior deep cervical group, while a single large vessel descends along the posterior border of the sternomastoid muscle to terminate in one of the inferior deep cervical nodes.

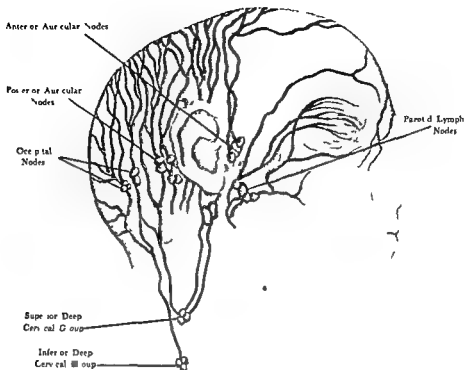


Fig 44—Lymphatic circulation of the scalp

INNERVATION

The skin of the scalp in front of the auricle is supplied by four cutaneous nerves viz, the medial part by the supratrochlear and the supraorbital branches of the ophthalmic division of the trigeminus, and the lateral part by the zygomaticotemporal branch of the maxillary division and the auriculotemporal branch of the mandibular division of the same nerve.

The portion of the scalp behind the auricle also receives four cutaneous nerves laterally it is supplied by the great auricular and small occipital branches of the cervical plexus which contain filaments from the second and third cervical nerves and medially it receives the great and smaller occipital nerves which are derived from the medial branches of the posterior primary divisions of the second and third cervical nerves respectively.

Nerve fibers appear in concentric circles about the lower two thirds of the hair follicle, with slender filaments appearing on the follicle wall and passing to the root sheath. Sweat and sebaceous glands are also individually supplied with a plexus of nerve fibers penetrating the membrana propria.

Free nerve endings occur around the sweat glands, in the papillae and root sheaths of the hair follicles and in the subepithelial and intermuscular connective

tissue and in serous membranes. After the nerve fibers have subdivided in the sub-epithelial connective tissue, losing their medullary and original sheaths, they continue as naked axis cylinders consisting of delicate filaments.

The axis cylinders in turn subdivide, forming primary and secondary plexuses from which many fibrillae penetrate the subepithelial basement membrane and multiply between the overspreading epithelial cells where they end in minute nodules or flattened discs. In the epidermis, the nerve fibrillae are limited to the stratum mucosum, but in the cornea they reach the surface layers of epithelium.

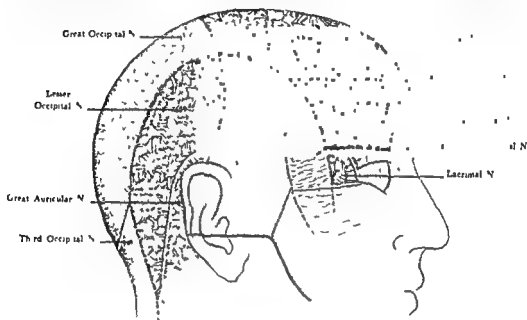


FIG. 45. Distribution of sensory nerves of the scalp.

MUSCULATURE

The muscles of the scalp are comparatively simple—they are the epicranicus and those of the auricle. The former is a muscle with two posterior and two anterior bellies and an intervening tendon called the galea aponeurotica which stretches uninterruptedly across the median plane of the cranium. This tendon is a continuous membrane which slips over the calvaria and has lateral attachments to the temporal ridge and behind between the posterior bellies, to the superior nuchal lines and the occipital bone. It is without any anterior osseous attachment.

Each posterior occipital belly has its inception as a broad, flat band appearing from the lateral two-thirds of the superior nuchal line of the occipital bone. Each anterior belly originates from the galea aponeurotica at the level of the coronal sutures with no bony attachments. This frontalis portion reaches downward to the supraorbital arch where it combines with the orbicularis oculi and corrugator supercilii muscles. It extends across the entire forehead blending in the median plane with the muscle of the other side.

The nerve supply is furnished by the posterior auricular branch of the facial nerve, while the frontalis is supplied by the temporal branches of the same nerve.

There are three auricular muscles originating outside the anatomical limits of the ear—the posterior, superior, and anterior, all elementary and normally functionless. The posterior is a narrow slip having its inception on the surface of the mastoid process and then being inserted into the cranial surface of the auricle, thus forming a bridge between the two which conceals the posterior auricular vessels and nerve. The superior is a smaller muscle which arises and fans out from the temporal fascia and then descends until it is inserted into the top of the root of the auricle. The anterior is another small muscle found in front of the superior, stretching obliquely between the temporal fascia and the uppermost portion of the root of the auricle.

The hairs of the scalp have their own bundles of nonstriped muscle fibers known as the erector pili. These muscles are connected with the follicles directly below the orifice of the sebaceous glands somewhere between the middle and upper third of the shaft. They take an oblique course from that point of their attachment to the papillary layer of the corium, to the tissue closely associated with the follicles. There may be a confluence of muscles serving a group of separate follicles, in which case some of the follicles have been found without a vestige of muscle. The hair follicles of the eyelashes, eyebrows, axillae, and the nose are not usually supplied with muscles.

As is indicated by their name, the action of the erector pili muscles is responsible for the erection of the hairs. Since the follicle normally lies at an angle to the skin's surface, the contraction of the muscle forces it to a new, right angle position which causes the hair to stand upright.

The pilomotor reflex may be caused by a drop in temperature when the muscles contract over the skin's surface to form what is commonly known as 'goose flesh'. It may also originate in emotional disturbances associated with anger or fear, a reaction so frequently observed in the alerted, bristling animal.

3. ARRANGEMENT PATTERNS AND REGIONAL DISTRIBUTION

Except for the palms, soles, parts of the dorsal aspects of the fingers and toes, portions of the external genital organs and the lips, a human being's entire body is covered with hair. The greatest percentage of these are soft, delicate secondary lanugo hairs called vellus which appear in the fourth or fifth month after birth. They are comparatively free from dark pigmentation and easily escape notice by the naked eye.

Transition and terminal hairs with well formed medullas account for the more obvious hirsute covering. It has been estimated by painstaking count that there are approximately one thousand hairs of all types per square inch of scalp. Since the average surface area of an adult's scalp supposedly encompasses 120 square inches, it requires 120 000 hairs to cover it. The finer the hair, the more numerous are the shafts present in a given area: blond hairs are usually finest and average 140,000, brown and black about 110 000, and the coarser red hair, 90,000.

The densest growth is in the region of the vertex where, measured by square centimeters there are 300 hairs. The occipital regions are less crowded with 200 per square centimeter and the beard and the mons veneris have only 30 or 40 per

square centimeter, with the back of the hand showing a distribution of merely 15 to 20 in the same space. Hair allowed to grow to its full length will measure between 22 and 27 inches and sometimes grow as long as 36 inches, but this is indeed rare.

The early lanugo hair of fetal life disappears at birth or shortly afterward to give place to the vellus which, in turn, through a slow evolutionary process of developing into darker larger, stiffer and coarse hair through several generations, finally evolves the hairs which are characterized as terminal. The follicle by becoming comparatively hypertrophied has adapted itself to the production of this type hair which covers 85 per cent of the scalp, the other 15 per cent still being occupied by vellus and transitional types.

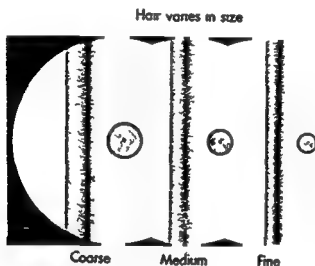


Fig. 46—Diagrammatic sketch illustrating the variation in size of the hair shaft. (Courtesy Mr. R. Reed and the Toni Company.)

Puberty and early adolescence are the periods of most rapid hair development. In the axillary and pubic hair regions the flattened somewhat irregular cuticular scaled hair shaft 3 to 9 cm. in length slightly smaller than the hair of the scalp accompany the growth of the sex glands of internal secretion. These hairs are irregularly twisted on their own axes which accounts for the familiar linking.

The distribution of pubic hair differs with sex. A woman's labia minora and the inner half of the labia majora are glabrous while analogous parts of the male anatomy are partly covered by hair. Men have patches of hair about the nipples, the middle of the chest along both sides of the umbilicus and in the deltoid and infra-scapular regions while only some females have hairs scattered about the nipples and the center of the chest.

Both sexes have a well-defined horizontal line at the meeting of the hypogastric and pubic regions which marks the border of the pubic hair. Both sexes also have a triangular pattern of softer hair belonging to the trunk whose apex is approximately at the umbilicus.

Both sexes have terminal hairs on the legs and a thick downy growth on the arms. A man's hair pattern usually extends from the ankle up to and covering most of the thigh while only the inner side of a woman's legs have a comparable hair growth and her thighs are completely clear.

The hair of the eyebrows or supercilia are terminal hairs of a particular crescent shape with pointed ends wider in proportion to their length than the average. They appear singly and under microscopic examination prove to have an angular cross section, a large medulla and flattened cuticular scales except at the end. From the age of 31 they change in character and in the male are replaced by curved or twisted hairs resembling those of the beard at times from $\frac{1}{2}$ to 10 cm in

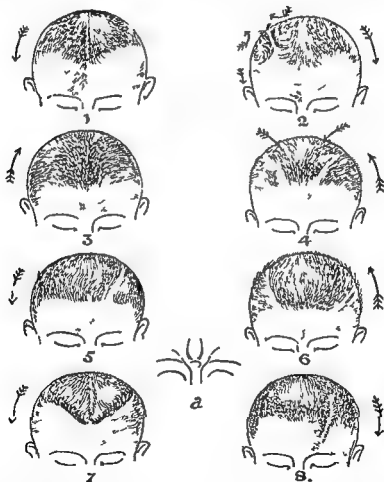


Fig. 47—Distribution of fetal hair. 1 Downward direction with undefined central part. 2 Central and lateral streams downward with upward feathering on right. 3 Upward direction on lower border with undefined part. 4 Central streams downward, lateral streams upward. 5 Downward direction on left parting. 6 Upward direction on right parting. 7 V shaped area downward direction with upturned borders. 8 Downward direction with left parting over forehead. (Drawings by Dr. K. L. L. courtesy Dr. W. J. O'Donovan, The Harter & A. Churchill, London.)

length. There is a definite hair pattern to the eyebrows even in fetal life, each possessing a short medial and long lateral portion. In the medial portion the hairs tend to turn upward and outward while in the outer section they take a downward and inward course. The eyelashes are composed of long curved single hairs like those of the early eyebrows but they remain so not changing in later life.

The hairs of the nose, the vibrissae are also of strong terminal type, curved scaly but ending bluntly rather than in points. The hairs appearing on the ears of some persons resemble the coarse twisted shafts of the post middle age eyebrows.

During interine life it is already apparent that hair follows various linear patterns. Some authorities believe that these groupings occur because the hairs originally emerged from under the free edge of a scalp pattern which no longer persists in most mammals. Others believe that the linear hair groupings are due to the distribution of the elastic tissue fibers of the cutis. We do know that the hair slope is the consequence of the follicles oblique angle to the surface of the skin and that what causes the follicles angularity of position is possibly due to muscular pull.

There are also authorities who claim that in addition to these anatomic factors the hair patterns are equally determined by external mechanical factors such as pressure friction and muscular traction. Beard growth patterns do not change but we are aware of the simplicity of altering scalp patterns in the young merely by changing the partings in the hair.

Fetal hair patterns repeat again and again the central left and right lateral partings which are reminiscent of ancestral methods of hair dressing and have nothing in common with anthropoid apes except for the central parting of the chimpanzee. Apes hair on the frontal region passes naturally from the region of the eyebrows directly over the low forehead and backward over the arch of the cranium in one continuous flow.

The hair whorls in the human being usually include a forward movement from the vertex to the brow and at the junction of the vertex many natural partings reveal themselves in several directions. It is possible that this is an atavism from the animal state when rain running off the creatures backs took certain well defined routes through their fur.

In man asymmetrical patterns are the rule. As shown by the chart which merely illustrates some of the possibilities extreme variability is characteristic of human hair patterns due in part to the many differing methods of hair combing brushing and dressing in addition to the peculiarities of growth and abnormalities of the cranium. This theory would indicate that hair patterns are another proof of the inheritance of acquired characteristics.

4 ANTHROPOLOGY*

Hair form and color have long provided man with a readily identifiable physical feature ideal for racial classification. Most classifications whether races were considered to be two or fifty two in number use this criterion to a lesser or greater degree. The macroscopic differences though showing a considerable amount of

*I am greatly indebted to M. Howard Malachuk for his valuable assistance in the preparation of the section on anthropology.

overlapping, are of greatest value. Investigators searching for more exact divisions have created the great bulk of the literature, but have not as yet completely clarified the situation.

GROSS HAIR FORM

Hair varies in form from straight hair through variations of curves, with three basic types of the latter being recognized

- 1 Wavy hair, which forms a two dimensional figure, that varies in linear distance between the crests of the waves
- 2 Helical hair, which forms a three dimensional figure, with loops of a constant diameter as far as each hair is concerned
- 3 Spiral hair, which is also three dimensional, but its loops diminish outward from the scalp in diameter, giving each hair a springlike appearance. The extreme form has narrow loops that intertwine in clusters with intervening hairless patches of scalp (peppercorn hair)

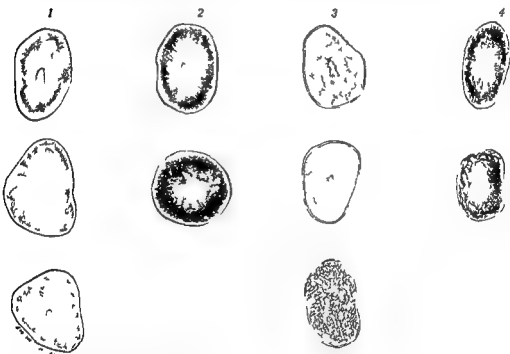


Fig 48 — Variations on single hair shafts. (After Kneberg)

- 1 Caucasoid (straight) a Index 68 b index 85 c index 90
- 2 Caucasoid (wavy) a Index 73 b index 100
- 3 Caucasoid (slightly wavy) a Index 67 b index 74 c index 57
- 4 Negro (kinky) a Index 56 b index 69

INHERITANCE

In the Negro, kinky hair seems to be dominant over Caucasian straight, wavy, or curly hair. Among Europeans, curly or wavy hair appears to be dominant or incompletely dominant over straight hair. The Mongoloid straight hair and the

straight hair of the American Indian seem to be dominant over the curly hair of the Caucasian. There is also considerable evidence of mutation in man, swine, and mice that might account for certain features of distribution, such as deeply waved and helical hair in Holland, Norway, and Ireland.

A quantitative study on five pairs of siblings has shown that in hair index and size, each pair showed similar trends with the most similarity occurring between identical twins.

DISTRIBUTION OF HAIR FORMS

Straight coarse hair is essentially a Mongoloid feature. Negroids tend to have the more curved forms while Caucasoids live in between forms and fine straight hair. As to the curved forms, an evolutionary selective value has been suggested as seen by its geographical location.

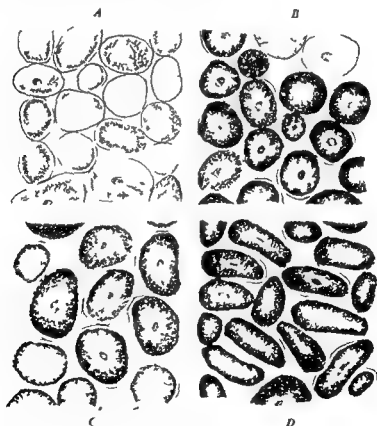


FIG. 49. Hair forms of four races. After Steggerda. Cross sections of hair from individuals of four racial groups showing far differences in shape and structure. A Dutch B Masa C Navajo D Negro.

Spiral hair is centered in Africa, or the heat equator, which suggests that its patches of bare scalp allow greater area for evaporation of sweat. This is seen foremost among the Bushmen of Kalahari, who live in the hottest most peppered (in

hair, as well as in the Pygmies of the African rain forest. Eastward, in Southern Arabia, tribesmen living south of the Ruba' al Khali desert have both spiral, helical, straight, and wavy forms. Spiral hair can be seen further spread in a few remote parts of the forest in the peninsula of India, in the Andaman Islands, in the forests of the Malay Peninsula, the inmost recesses of Sumatra, and in marginal areas of the Philippines.

Helical hair is centered in Africa and Melanesia, with islands of spiral haired Negritos in between. At both extremes, the movement of helical hair can be seen to be distributed to the north. It can be found among European and European like populations, but in these groups it is not found on the body, as with the Bushmen, Pygmies, and Negroes. At the other extreme, it is seen to occur among the Okinawans and some Japanese. This helical hair, when it forms a matlike shape, produces a dead air chamber which might be of protective value in the desert.

However, as many of us do well without any hair, the subject of functional values remains very confusing and a great mystery.

COLOR

Color depends not only upon pigmentation but also upon thickness of the shaft. Several other factors such as oil content and air content might also be involved. This must be taken into account when recording hair color values for young age groups. Several authors have offered quantitative data to substantiate this darkening of hair with age, which correlates with an increase in size of hair during the formative years. Other factors may also be involved in this color change. A longitudinal series of hairs from all Dutch children in Holland, Michigan, showed that the head hair became darker by almost one unit of the Fischer Saller scale with each year of age from 11 to 16. No significant sex differences were noted.

Distribution of Hair Color

Light hair is not confined only to northwestern Europe, but can be found in several tribes of Central Australia, where most of the children are blond but darken later, as well as in parts of North Africa, the Atlas Mountains, and the Near East. A high occurrence of light hair, or secondary European focus, is to be found in the Baltic countries. Red heads also appear occasionally among Negritos, Mongoloids, and South Australians. However, red hair and brown hair are commonest among the blue eyed, light skinned peoples of northwestern Europe and Britain.

CUTICULAR SCALES AND THE MEDULLA

It has been shown that the cuticular scales and the medullas of human head hair are related as are the same elements in the hair of the infrahuman mammals, to the diameters of the hair shafts to which they belong. These elements then, cannot be regarded as characteristic of race. Human head hairs have not been found to show any but one type of scale nor any but the continuous and the varieties of fragmental medullas. These elements do not show any of the characteristic differences of form which have been interpreted as being group differences among the infrahuman mammals.

The scales of the human hair shaft fall into the flattened type category and, like the scales of the infrahominid hairs, vary, in a general way, inversely with the diameter of the shaft. The scale forms, therefore, are unrelated to race. It was found that the scales varied on contemporaneous hairs from the same herd, when the diameters varied. Moreover, differences exist on the same hair from top to bottom. Generally, the coarser the hair, the finer the scale.

Age studies have verified these facts. It was also shown that the morphologic type of medulla is not determined by age but by shaft diameter. The relative sizes of the cuticular scales bear no relationship to age, but vary inversely with the diameter of the hair shaft.

The increase in hair size or area during youth would lead one to expect from the above facts a change in medulla incidence. This has been borne out. The percentage incidence for medullas is very low at birth and increases rapidly during the first seven months of life. After seven months extending to the second year there is a decrease followed by great irregularity but with a general tendency to rise.

One would also suggest a similar change in the scale count. This also has been borne out. The scale count drops slightly during the first year for most subjects.

CROSS SECTIONS

It is in this field that the first endeavor was made to find absolute criteria in racial hair groups. A history of the early literature shows numerous claims to this effect. Generally speaking however it can be said that Mongoloid hair is more nearly circular in cross section. Negroid hair elliptical and Caucasoid intermediary.

This though is far from absolute. The size and shape of cross section of individual hairs from the same person may vary widely. With a modification of the Hardy technique of sectioning it is now possible to treat large samples of hair statistically and thus arrive at a more valid estimate of this criterion.

This method has the advantage over the old embedding and microtome technique in that as many as 150 to 200 hairs may be sectioned at once with the time required for making a permanent slide containing this number being as little as 10 minutes as compared with 2 or 3 days in the older technique.

The device is a small metal plate 3 inches long 1 inch wide and 1/8 inch thick. It has a narrow slot 3/8 inch long into which can be packed 150 to 200 hairs depending upon the thickness of the hairs and the degree of tightness with which they are packed. The hairs are washed in carbon tetrachloride dried and packed into the slot. Then they are cut off even with the metal plate on both the top and bottom sides of the plate. They are then pushed partly through the fiber slot by means of a metal screw. Thus by turning this screw the thickness of cross section can be regulated. The layer of protruding hair tips is then coated with a thin solution of celluloid (11 Gm celluloid 40 cc acetone and 20 cc amyl acetate). When the solution hardens this layer is cut off with a sharp razor blade and the resulting strip mounted in Euparal or Canada balsam.

Garn has developed two techniques that enable the microscopist to obtain serial sections of undistorted hair with each hair identifiable in each section. The

first method is suitable for paraffin embedding, and the second method allows for the rapid production of thicker sections

The first method uses a cardboard support and employs paraffin melting at 62°C to hold the hair securely. After the hairs are selected from a single lock, washed in solvent, and the dirt is removed, they are fixed to the support with globules of cement. It is important to fix them in place without distorting the wave form and also to space them properly. Indexing hairs of larger diameter, bent parallel to the human hairs, are set at either side. After soaking the finished support in xylol, embedding is completed without dehydration. The paraffin block is

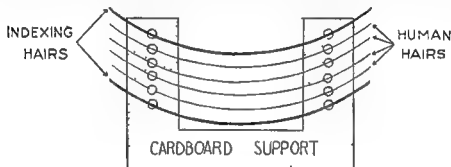


Fig 50—Method for obtaining serial sections of undistorted hair, and suitable for paraffin embedding (After Garn)

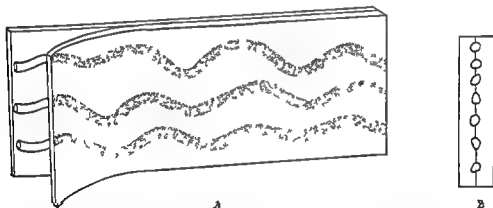


Fig 51—A more rapid method but a less perfect lamination technique for the production of thicker sections of undistorted hair (After Garn) A, Cellulose acetate strip 0.030 cm thick. Hairs aligned parallel B Typical cross section

cut from the hardened paraffin in the usual manner, the curvature of the enclosed hairs marked on the outside, and the sections made. During sectioning the object carrier of the microtome is readjusted every few rotations so that the sections are perpendicular to the hairs. Sections 15 to 29 microns thick cut well. The paraffin sections are floated on albumin coated slides, dried and deparaffined. The large fibers at the ends of each section aid in location under the microscope and serve to

indicate sections that have been inverted or reverted during handling. By its constant relative position each hair can be identified in successive sections. The optical activity of the hairs, if any, can be studied in sections so made.

The second method is a rapid but less perfect lamination technique. The hairs are oriented on a cellulose acetate strip tacked down with acetone and covered with a second acetate sheet. Acetone or a suitable cement may be used for laminating and moderate heat and a few pounds of pressure help the contact. Within 10 or 15 minutes sections can be made either freehand or with the aid of a jig. Again the sections must be perpendicular to the hairs rather than to the lamination. This technique facilitates rapid study of cross section form, area and rotation about the longitudinal axis. Sections should be made within a few days after laminating.

HAIR INDEX

The hair index is a numerical expression of the shape of hair. Assuming that the outline of the cross section of the shaft is regular and symmetrical it is derived from the following formula:

$$\text{index} = \frac{\text{least diameter} \times 100}{\text{greatest diameter}}$$

The index drops sharply during the first two years of life after which there is irregularity with little consistency in trend. In other words the cross section of hair as expressed by index changes from a more nearly round to a more nearly oval shape in the first two years of life.

It has also been shown that among certain races the males have a higher index than the females and the index of the latter tends to be more variable.

The same conditions beset the use of index as a reliable criterion as does the use of cross sections of hair. It appears that hair form and the shape of the cross section of the hair shaft are not interdependent and the form cannot be predicted from the shape of the cross section. Perfectly straight hair may be flat and deeply curved hairs may be round.

AREAS

Assuming the outline of the cross section of the hair shaft is regular and symmetrical the area of cross section is taken to be

$$\text{Area} = \frac{1}{2} \text{ greatest diameter} \times \frac{1}{2} \text{ least diameter} \times 3.1416$$

It has been shown that the size or area shows a rapid and uniform increase during the first three or four years of life, a less rapid and less uniform increase the following six years and then either a slow increase or leveling off.

A correlation has been found between area of cross sectional surface and the weight of a given length of hair.

HAIR WEIGHT

Two decades ago that did not determine the

investigators looked for delicate instruments sections of hair

reliable criterion of racial hair flex test is employed to weight of hair use of

By weighing equi-length strands of hair on a fine Trommer balance sensitive to a hundredth of a milligram, it was found possible to separate the hairs of the three grand divisions without difficulty. In this study, the average difference between male Negroes and male Caucasoids was 15 per cent, and between male Caucasoids and male Mongoloids there was a difference of 60 per cent. Within the Caucasoid grand division, the hair of Nordics weighed least, that of the Alpines was heavier, and that of the Mediterraneans was heaviest. Those of mixed race showed a tendency to vary between the two groups from which they came. The average weight for males in the Caucasoid group was found to be 18 per cent greater than for females, which was found to be approximately the same in the subdivisions of the Caucasoid group. Very little overlapping was noticed between the races.

Microscopic study of the cross sections of these hairs indicated that differences in area of cross section, pigmentation, and the appearance of "air bubbles" were the variable factors. The latter two factors were repudiated in another study. However, the difference in diameter of cross section between the sexes averages 10 per cent, which is a difference of 20 per cent in cross section. Thus, this is well in agreement with the 18 per cent difference found between the sexes.

FACIAL HAIR

No racial or sexual differences have been noted in the actual numbers of facial hairs in a study of Negroes and whites. The facial hairs of women of the white race slightly exceed those of the Negro race in length. As can also be seen by the naked eye, this study showed that after the tenth year the diameter of the facial hair in the male greatly exceeds the diameter in the female, but the smaller hairs (the lanugo) in comparable regions of the face are the same in both sexes throughout life. Also it was seen that the facial hairs of the white race show a greater average thickness than the Negro race.

Mongoloid men have fewer hairs on their face than European men. A Tungus or an Eskimo will have one fourth to one eighth as many active beard follicles as a European, and the beard grows one fourth as fast and has to be shaved one fourth as often. This has been suggested as of selective value, as long beards in the Arctic cause the breath moisture to freeze, and hence the face freezes underneath too.

PILOMETRIC MEASUREMENTS

Comparatively little has been done in the field of pilometric measurements in any of the various possibilities that it offers. As a cursory comment, in one of the earlier reports concerning a pilometric machine of reliable accuracy, a racial difference was suggested, though the size of the sample prevented the statement from being at all conclusive. This study showed a group of 42 healthy white male and female adults to have values for firmness of attachment ranging between 22 and 60 grams with an average of 34 grams. The tensile strength varied between 65 and 120 grams averaging 95 grams. In the four male adult Chinese examined the firmness of attachment varied between 64 and 88 grams and the tensile strength between 141 and 165 grams. The individuals of the Chinese group might, of course, be of an exceptional nature, but the values obtained indicate another approach that might be of worth both to the medical investigator and the student of race.

Maibach recently studied a fair sized sample of Japanese and Caucasian hair from which a more exact evaluation of these factors has ensued. Using an extensometer of the electric strain gauge type, he showed that the force required to break a fiber was found to be directly proportional to the size of the fiber, and, similarly, tensile strength was found not to vary significantly between the two racial groups. Generally speaking it would seem that the racial differences are quantitative rather than qualitative. A great deal more remains to be done with this type of testing which is still relatively unexplored before positive conclusions can be drawn.

He also found that certain physical properties of head hair have race and sex differences. Using the objective criterion of comparison of unit weight male samples of Japanese head hair were found to be more coarse than female samples and Japanese samples were found to be more coarse than Caucasian samples. At this point significant differences end.

ALOPECIA

A recent study showed that alopecia was less common to a very considerable degree among Chinese men than among Caucasian men. When baldness was present, it occurred at a later age in the Chinese men. It was shown with this sample group that the Chinese men had a frequency of absence of bilateral recessions along the anterior border of the hair line in the frontoparietal regions that equaled and favorably compared with that seen among Caucasian women.

It is generally believed that white people are the most susceptible to baldness of the male pattern type with the highest predominance being in eastern Mediterranean stock. The Negro groups have less baldness and the Mongolian peoples the least. This is in direct contrast with the amount of facial and body hair as Caucasians have the most, Mongolians have the least and Negroids are intermediate.

VARIATION

It can be quite clearly seen that there is not only racial variation but probably to an even greater extent there is variation among the hairs on one head. Single hair shafts show an increase in size as they progress from the scalp. Single hairs vary in and vary in their various parts as do hairs that lie side by side on the same head. Cross sections are not only round, oval and elliptical but also triangular, prismatic and kidney shaped. Hair growth is furthermore dependent on age, health, diet and glandular balance to mention but a few of the factors.

For this reason any future work that is to be done in this field will have to employ a method that will allow for large samples and statistical treatment. Only in this way will a valid set of useful criteria be found.

5. PHYSIOLOGY

PIGMENTATION

The color of the hair is determined in part by racial and hereditary factors and its appearance to the observer is due to the amount of pigment present, its oxidation stage and its arrangement in the hair cells. All of these contributing factors probably occur in the hair bulb itself, since pigment is not carried into a

fading due to exposure. No circulating process is present to disturb pigmentation once it is determined, and gray or white hairs appear as new replacements during hair exchange. One's hair does not 'turn' white. What happens is that pigmented hair shafts are shed and their successors from the same follicle are without pigmentation granules or possess them in an undeveloped or unoxidized form, the unknown pigment producing stimulant having been lost.

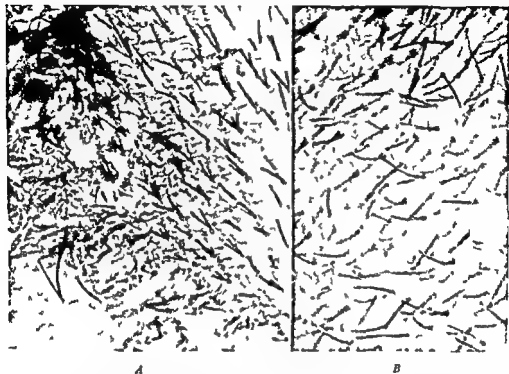


Fig 52—A and B Two hairs growing in a single follicle. This phenomenon is not uncommon and serial sections from any scalp may show similar instances. (Courtesy Dr L. Loewenthal and J. Invest. Dermat.)

The cortical cells containing pigment were originally basal cells of the living epithelium of the hair. Experiments with dopa oxydase proved that these are the sole pigment producing cells. Color does not originate in the papilla since its connective tissue cells have a negative reaction to oxydase as do all cells not of epithelial origin. Therefore one of the functions of the papillary cells must be considered the storage of the pigment and they are classed as chromatophores having no part in the actual manufacture of the pigment. The pigment granules are produced *in situ* by the cells of the hair bulb itself. As these develop, they undergo further differentiation and become the cortical cells continuing to produce pigment. As the hair shafts, composed largely of cortical cells grow upward it is their pigment content which imparts the characteristic color to the hair.

There are two pigments involved in hair coloration—granules of brown pigment forming the group of acid insoluble melanin and the more diffuse red pigment of the cortex which is an acid soluble melanoprotein. Either or both pigments may

be present in a given hair, but the nongranular type is less frequent. Brown hair results from a mixture of melanin and melanoprotein while black hair, as we know it, is merely a deeper concentration of brown and the nongranular pigment accounts for nil. It is the consensus that different colors represent different stages in the oxidation of a single pigment base and the shade indicates the stage of oxidation of this pigment. Danforth believes that it is at this point that the hereditary factors count, regulating as they must, the precise amount of oxidation that shall take place to produce each hair color.

The melanin pigments are diffusely distributed in light colored hair. In dark hair, they are found in granular form. Their chemical nature is not precisely known as yet. According to Giroud and Leblond, there seems to be a chromatophore component melanine, united to amino acids, polypeptides or proteins. The presence of iron is in doubt, except in the pigments of red hair, as described by Rothman and Flesch.

After an iron containing pigment was isolated from human red hair, and not found in hair of other colors the question arose as to whether the total amount of iron in red hair is relatively greater, reflecting the presence of an additional iron compound in red hair. Dutcher and Rothman could only find data on the iron content of human hair in the literature of the last century. According to the analyses of Baudrimont the iron content in the ash of hair is as follows: brown, 10.9 per cent; red, 9.7 per cent; white, 8.4 per cent; black, 8.1 per cent, and blonde, 4.2 per cent of the ash expressed in Fe_2O_3 . Dutcher and Rothman state that red hair contains greater amounts of iron (9.78 mg per cent) than hair of any other shade. The presence of an iron pigment in red hair does not account for the whole difference. It is assumed that in the hair iron is split off from the organic pigment complex. They also observed that the ash content of red hair is significantly higher than that of hair of other colors.

Few persons have hairs of an identical color over their entire scalp. Detailed examination under various lights shows that many shades of hair occur on one head. Blondes in particular sometimes reveal hundreds of varying shades on a single normal scalp.

Depigmented hairs both gray and white are according to Danforth of two hereditary types: dominant and recessive or albinotic. Whiteness due to the latter is believed to be dependent on the inhibition of certain heritable factors since albino animals correspond to all the recognized color classes except that they lack the ability to develop their color potential. Albinism represents a recessive hereditary trait since albinos may have had normal parents and do not invariably give birth to albinos.

Canities is the term designating the graying or whitening of the hair over the entire scalp while *polio* describes blanching which occurs in specified local areas and is a dominant trait appearing through successive generations. Racial hereditary and individual factors all contribute to the widest possible variations in the dominant types of nonpigmented hairs. The chemical mechanism which takes place in the hair bulb just before the hair loses its pigment and becomes white is unknown. There is no absolute age level at which these hairs appear and their

blanching is totally unrelated to the aging of other tissues but may have something to do with the enclosure system and its nourishment

In ordinary graying the loss of pigment is gradual and the hairs seem dotted with gray and varying shades of the original color before the exchange produces an all white hair. In canities, the hair never returns to its original color, there can be no recovery. Both in alopecia areata and hair situated on a patch of vitiligo, the exchange eventually, upon cure, returns hair with its normal pigment. Evidently these two diseases only temporarily inhibit the formation and deposit of pigment in the cortical cells of the hair, while canities has a permanent effect

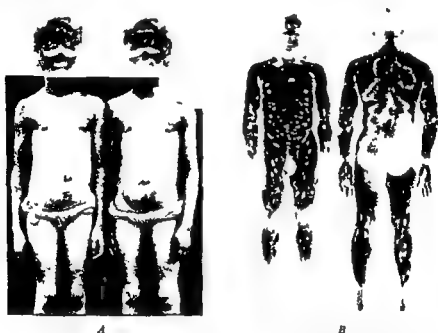


Fig 53—A Negro twins, each of whom is albino (Courtesy Sutton and Sutton Diseases of the Skin Original Photograph by Drs Wakefield and S Dellinger)

B Prebald albinism The leopard man A dominant, hereditary, pigmentary anomaly (Courtesy Sutton and Sutton Diseases of the Skin Original photograph by Dr C Keeler)

Premature canities, according to McCarthy, may begin in childhood with a scattering of unnoticeable hairs on one side of the head. By adolescence, the numbers have increased sufficiently to be obvious to the observer. By 25 or 30, this abnormal canities has already produced a salt and pepper look, and by 40 or 45 the patient is completely gray. Contrasted with this, normal graying begins at 35 or thereabouts with a group of nonpigmented hairs in the temporal regions which increase normally. It is possible that after these areas have whitened, the rest of the scalp may be spared or several areas may become white simultaneously

Frost points out the fact that graying hair has been induced in all species of animals studied and has been observed in human infants under a variety of dietary

deficiency conditions which would seem to indicate that graying especially during growth is a characteristic manifestation of certain types of dietary deficiency. The intent in the great majority of experiments has been to develop a specific deficiency of only one pigmentary factor. There is little chance that such extreme specific dietary imbalances often occur among human beings but multiple deficiencies undoubtedly do. Several years ago a great furor was aroused by the observation that the vitamin B complex contained several different antigay hair factors. These reports were based on the fact that when young rats were fed a purified diet low in the vitamin B complex and deficient in para aminobenzoic acid and pantothenic



Fig. 54. Partial hereditary pancytopenia in an 11-year-old girl associated with an underlying atrophy of the scalp and elsewhere on the body. Only sister and no other is similarly affected. (Courtesy McCarthy. *11* *Seases of the Hair*.)

acid growth was retarded and cutaneous changes were observed. These changes consisted of dermatitis, diffuse alopecia, and symmetrical pattern graying of the fur. When the diet was supplemented by the addition of pantothenic acid and para aminobenzoic acid, growth was promoted and the generalized dermatitis and symmetrical pattern grayness disappeared. A morphologic explanation for the graying of the fur of rats on pantothenic acid deficiency diets was advanced by Ralli and Graef, who demonstrated that the sudden graying was the result of atrophy of the

hair bulbs and follicles, due to cessation of melanin deposition in tissues. One of these investigators also observed that graying of the fur occurs much earlier in animals on a low salt intake, and that adrenalectomy will bring about an increase in the deposition of melanin in the hair bulbs of rats graying as a result of pantothenic acid deficiency.

Despite the allegations of early investigators in this field, until recently there was no clinical evidence in human beings that either pantothenic acid or para-aminobenzoic acid could either convert the gray color of the hair to black, restore the pigmentation in vitiligo, or induce depigmentation of lentigines. As has been mentioned elsewhere, it is a mistake to assume that findings in animal experimentation apply to conditions in man. It is true that a great deal can be learned about the effects of various nutritional agents on other parts of the human body from experiments on lower animals. However, the human skin differs so profoundly from

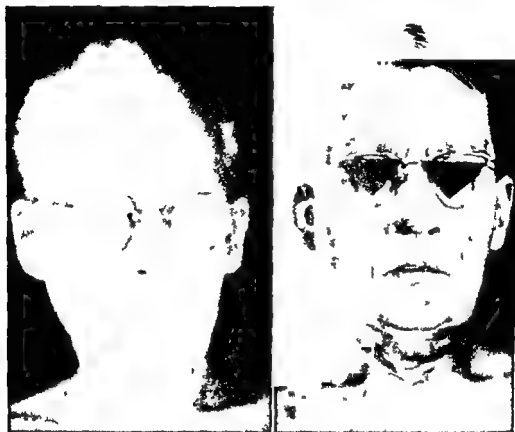


Fig. 55—Vitiligo of forehead with graying of hairs in involved region. (Courtesy Dr. Samuel Peck.)

that of other animals in its morphology and physiology, and in the way in which it reacts to therapy, that we can learn about it only from observations and investigations performed on human beings as in the recent report by Zarafonitis. He noted that darkening of gray hair may result from prolonged administration of para aminobenzoic acid. His subjects had received from 6 to 24 Gm. of para aminobenzoic acid daily for the treatment of various primary cutaneous disorders. The effect on hair color was first noted six months after para aminobenzoic acid therapy was begun in one case, but a longer interval was usually required to bring about the change. The effect was not uniform, however, since other patients, who received para aminobenzoic acid for comparable periods failed to exhibit significant change in hair color. There appeared to be no correlation between the darkening effect

and the duration of grayness prior to treatment. The color alteration was observed to occur during the administration of para-aminobenzoic acid alone. Supplemental vitamin therapy appeared not to influence the effect on hair color in these subjects.

It should be noted that these patients were given large quantities of para-aminobenzoic acid in order to control their disease processes. The amounts used therefore, were far greater and of higher potential toxicity than those employed by others



A

B

FIG. 56. A Patient with lye phollastoma cutis and gray hair. B Same patient with darkening of the hair after six months of therapy with para-aminobenzoic acid. (Courtesy Dr. C. J. D. Zarafonitis and J. Invest. Dermat.)

who were deliberately seeking to alter gray hair. The mechanism by which para-aminobenzoic acid may cause repigmentation of gray hair in certain individuals remains obscure. Further study of the metabolic activities of para-aminobenzoic acid may aid in clarifying the pigmentation process. In this connection Zarafonitis noted that in scleroderma and dermatitis herpetiformis the associated cutaneous hyperpigmentation usually fades to a lighter, more natural coloration during para-aminobenzoic acid therapy.

Frost maintains that it has never been proved that graying has ever actually been induced under ordinary conditions by mild chronic deficiencies of various B

complex and mineral elements. Since genetic, hormonal, and dietary influences have all been shown to play a part in pigmentary processes, the etiology of graying in individual people is unavoidably obscure. He, too, believes that the practice of ascribing human curative or prophylactic properties to single pigmentation factors which have been found effective in animals under highly specialized and controlled conditions is not well founded. It is uncertain that graying in adult human beings is ever caused by a dietary deficiency and, if such were the case, the deficiency would surely involve more than one of the factors thus far shown to be necessary for normal pigmentation processes in animals.

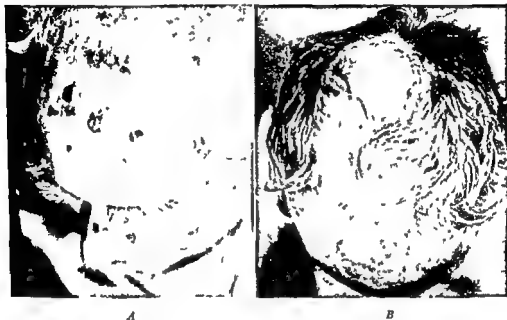


Fig 57—A Patient with dermatitis herpetiformis and gray hair. B Same patient with darkening of the hair after six months of therapy with para-aminobenzoic acid. (Courtesy Dr C J D Zarafonitis and J Invest Dermat)

Because of genetic influences, graying in adults is not considered a manifestation of unnatural physiologic change. The fact that graying occurs in children on extremely poor diets is of considerable interest but does not contribute toward assessing the situation with regard to adults. Experiments with human beings were carried out before the need for pteroylglutamic acid for normal pigmentation was established in animals. Most of the human studies involved the use of pantothenic acid. Now it appears that there may be a physiologic relationship between these two factors. The apparent functional relationship between pteroylglutamic acid and the antipernicious anemia principle of liver seems to Frost to be of noteworthy interest, particularly since the liver extract has been reputed to have an effect on graying in isolated cases. When more is known about the nutritional role of the various pigmentogenetic factors and about their interactions in essential enzyme systems, further study of their effects on graying in human beings may be indicated.

Goldman recently observed several cases of achroniotrichia and hypotrichosis in children due to intestinal parasitism and also following sulfonamide therapy. These disturbances improved following Panthenol (alcohol of pantothenic acid) therapy in doses of 0.5 to 1.5 Gm daily over a period of one to three months.

Neurotrophic causes may produce changes in hair color. Premature graying of the hair has been occasionally observed following illnesses of various kinds as well as following certain physical and mental trauma. In addition to severe general illnesses such as typhus, malaria, and influenza being followed by canities, certain disturbances of the nervous system may play an influential role. Neuralgia of the facial muscles and traumatic ophthalmia with optic neuritis have had subsequent canities in their wake. Frodin-Bonn stated that other examples of graying could be found due to injuries and illness involving the central nervous system, and he considered premature graying due to some form of disturbance of that system.



Fig. 100 Localized graying of the hair following herpes zoster of the supraorbital nerve (Courtesy McCarthy, *Diseases of the Hair*)

Galewski reported: "In general all severe illnesses and disturbances of the general well being can under certain conditions produce a premature lasting or temporary whitening of the hair. The patient however must have a definite predisposition, quite often hereditary; otherwise premature canities would be observed more frequently than it actually is."

White forelock may be considered a physiologic pattern abnormality of the skin and its appendages. Cockayne has studied this exhaustively and he divides all such persons into two types: the piebald and those with a white forelock only. The

former show a number of unpigmented areas of skin (leucoderma) as well as the forelock. In both types the abnormality is present at birth and is a dominant hereditary trait.

Frohn Bonn studied several cases of localized canities and discovered that some of them had a nevus or tumorous character. In one, for example, a combined infectious and nevus lesion was present in the central part of the hair discoloration. Many years before, Schein described such tumors with white hair, observing in addition that these blanched hairs grew more rapidly than the normal surrounding hair. In explaining their etiology, Schein worked from the premise that the quantitative color development is a local characteristic and the unusual speed of growth and exchange used up the normal pigment allotment. When a pigmented nevus is the root of the trouble, such a great amount of pigment goes into the nevus cell, he believed, that no color is left for its appendages, and thus he accounted for the whitening of the hair in the area. According to Schein, the loss of pigment in such hair is based upon the exhaustion of pigment through increased use by adjoining tissue. In these cases, hereditary factors seem to play little or no part. In only one instance was it demonstrated that the same case of hair anomaly also existed in the patient's mother since infancy.



Fig. 59—Extensive poliosis in a 19 year-old girl. (Courtesy Dr. W. J. O'Donovan. *The Hair*, J. & A. Churchill, London.)

The deviation in pigmentation known as *pili annulati*, *leucotrichia annularis*, or ringed hair is an abnormality of the hair shaft which is revealed as spindle shaped white zones about $\frac{1}{5}$ inch in length, alternating with normally pigmented areas about $\frac{1}{50}$ inch in length. The size of the stripes varies somewhat with the length of the hair itself. When seen in reflected light, ringed hair appears as alternating, very narrow, almost ringlike, pigmented and white bands or zones. The diameter

of the hair shaft is not modified as in monilethrix and trichorrhexis nodosa. All hairs of the scalp or only a very few of them may be affected. Again a hair may be involved for only part of its length, the alternate zoning may begin at either the proximal or the distal end.

The origin of this banding of alternate light and dark color imparted to the involved hair is still in dispute. Older writers believed the white areas represented regions of pigment deficiency or atrophy probably as the result of intermittent periods of malnutrition of the hair papilla. This seemed to be borne out by the fact that ringed hair occasionally occurred in those persons who had just passed through a severe debilitating disease. Pincus has likened the changes in these hairs to analogous changes in nails (Beau's lines) which show intermittent and irregular periods of growth. He found pigment was also present in the white parts of the alternating bands but this pigment was obscured and rendered invisible by the presence of air bubbles between the cells of the outer cortical layers. These air bubbles prevented light from reaching the pigment in the depth of the hair shaft and, accordingly, their reflection of the rays of light imparted a white appearance to the affected portion of hair.

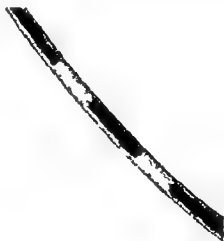


Fig. 60.—Ringed hair showing alternating light and dark bands.

Cady and Frotter carried out a very comprehensive and scientific study of this disease. Following Pincus' observations they proved the white color was due to a gas contained in the cortex of the hair. As soon as they removed the contained gas, no difference in color could be seen in the regions which had appeared white and those which had appeared dark. They concluded that ringed hair is therefore simply normally pigmented hair in which there are more or less regular accumulations of gas in both the cortex and medulla. Viewed in reflected light these regions appear light while in transmitted light they appear dark. Upon microscopic examination they found that the banded portions were primarily due to modification of the cortex and not to changes in or swellings of the medulla. The modified areas were seen as irregular spots extending for variable distances up and down the hair and sometimes nearly across the hair. Some of the modified areas were

unilateral. No explanation has ever been given as to how the gas gets into the hair. Cady and Trotter believe the gas is probably carbon dioxide, as it was completely dissolved out of the hair by a 5 per cent solution of sodium hydroxide which was fully saturated with air. It is this gas which clouds the outer layer of the cortex and thus conceals the pigment from view.



Fig 31 —Ringed hair. The three figures are all from the same hair. The one on the left depicts ringed hair via reflected light in the middle, via transmitted light and on the right, after rapid immersion in sodium hydroxide. (Courtesy McCarthy. Diseases of the Hair. Original photograph by Danforth. Hair, A. M. A. Press.)

RHEOLOGIC PROPERTIES

Combing, brushing, pinning and all types of curling exert strains on the hair shaft day after day and yet do not normally result in breakage. This phenomenon is explained by the elasticity inherent in each hair. Early in the present century,

experiments were carried out for the purpose of ascertaining how far hair could be stretched without damage whether it would resume its original length after relaxing the strain, and whether various chemicals affected elasticity

Lestwich in an attempt to answer these questions, tested the strength of a single hair by pulling it firmly and as evenly as possible over both sides of a Salter's letter balance noting the weight registered at that precise moment when the hair broke. Seven ounces seemed to be the maximum weight before permanent damage.

Then to determine whether curling irons used before testing would weaken the hair, Lestwich discovered this decreased the tensile strength of the individual hair by one half to one ounce. Since he neglected to note the degree of heat employed little of lasting value may be deduced from this experiment.

When Savill later attempted to check these previous findings she found a set of wholly contradictory results by frequent testing of permanently waved and natural hairs. The only deduction possible was a statement after experimentation that waved hair generally broke more easily than the natural even when the former had been immersed in oil for an hour before testing.

If nothing else these initial attempts with their crude mechanics proved by their indeterminate results that there was a necessity for the development of a machine which would with the greatest possible accuracy, be able to measure the rheologic properties of the hair such as its yield value elasticity, and tensile strength. An instrument was first devised by Copley to study the firmness of the attachment of hair to the scalp under certain chemical conditions. It consisted of a small clamp a hook and a ball bearing pulley fastened to a vertical stand. The clamp was designed to hold the hair and was fastened to a silk cord which passed over the pulley. A lusteroid tube of 100 cc. capacity was hung on the hook attached to the other end of the cord serving as a container for weights in grams of water. Clamp cord and the empty tube had a total weight of 8 grams.

This instrument was followed by an unproved machine called a pilometer based on the same principle but substituting a balance for the tube. It was also equipped to allow the measurement of a hair's elasticity and tensile strength. Experiments on this device indicate that Lestwich's findings were twice the actual tensile strength expectation of the average hair tested more accurately.

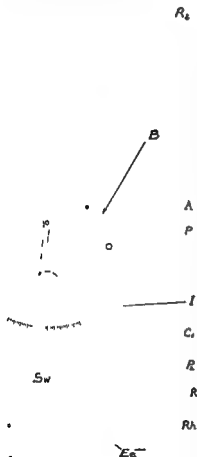
Copley reported that in a group of 42 healthy white male and female adults the firmness of attachment of the hair to the scalp ranged between 22 and 60 grams averaging 45 grams. Copley observed that the values should be amplified by a much larger number of observations in different male and female racial and age groups on healthy subjects and on hair from various parts of the scalp as well as other regions of the body.

For study of the tensile properties of hair there is also in recent use an extensometer of the electric strain gauge type. This instrument consists of three basic elements

1. A force sensitive unit producing an electrical signal proportional to the applied load. This unit is a type GI 8 120 Statham transducer which will indicate the magnitude of a tension or compression load ranging from zero to eight ounces.

- 2 A recording unit which measures and plots the magnitude of the electrical signal against time This recorder unit is a modified Brown High Speed Electronic Potentiometer
- 3 A constant rate of elongation device, adapted from a standard 6" lathe Six speeds are available for rate of elongation

In essence, the machine stretches a fiber at a constant rate and simultaneously a stress strain curve, one axis of the plot being the force applied to the fiber, the other representing the elongation produced in the fiber by this force



The balance, B, moves on two parallel, vertical rods, R_1 , R_2 , mounted on a base, E. A spring, Sp, to control the movement of the balance, is placed on rod R_2 between the base and the balance. The balance consists of the pendulum, PU, swinging on a plate, PL. The pendulum carries any given weight at W, and has a projection which serves as the indicator, I. Two pulleys, P_1 , P_2 are provided to lower the balance, one being fastened to the balance, and the other to an elevation, Ee, on the base. One end of a cord, C_1 , is attached on the elevation passes up and over pulley P_1 and returns to pulley P_2 , which rotates on its axis a small rod, SR. A third pulley, P_3 , on ball bearings, L, is vertically movable on rod R_1 by means of slide S, which can be affixed by a head screw, Sc to any desired height. The balance, B is pulled slowly downward without vibrations by means of the rotating handle, Rh. One end of another cord, C_2 is attached to the pendulum of the balance and the other end to the clamp, CL₁, which holds the hair, H while attached to the skin or after its detachment.

For measurements of properties of an isolated strand of hair, clamp CL₁ serves to hold one end, and clamp CL₂, at the base of the instrument, the other. A vertical scale, Sm, is used to measure the length of the strand of hair.

The instrument is operated as follows. A strand of hair still attached to the skin is clamped in CL₁, and the rotating handle is turned slowly until the hair becomes detached. The stress applied to the hair increases while the indicator moves along the scale, Sw, simultaneously with a pointer, Po, which is mounted behind the indicator and which remains at the maximum position after the hair becomes detached. There upon the indicator returns to zero. By pushing a button, A, the pointer can be brought back to zero.

The detached end of the hair is now put into clamp CL₂, and the rotating handle is again operated, but now for the purpose of measuring elasticity, yield value, and tensile strength.

62—Diagram of pilometer with attached explanation (Courtesy Dr Albert L Gopley)

Various manipulative procedures are possible. For example. The fiber may be stretched to break, giving a complete stress strain curve and allowing measurement of various features of this curve (initial slope, related to elastic modulus, yield and breaking points, breaking strength, per cent elongation at break). Either factor,

elongation or load, may be held constant and a study made of the change with time of the other factor. The return stress-strain curve may also be studied, obtained by allowing the fiber to shrink at a constant rate from a previous extension.

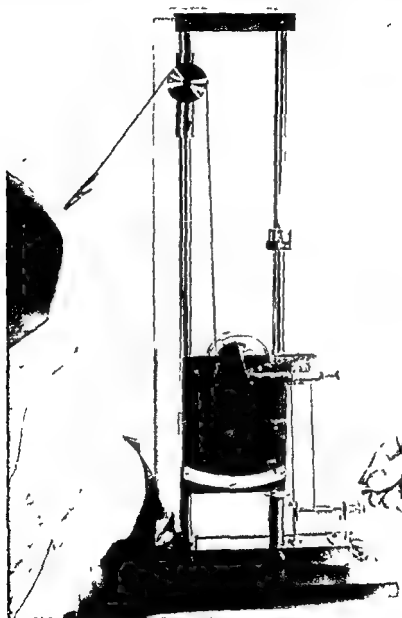


Fig. 63 Pilometer in actual operation. (Courtesy Dr. A. I. Copley, with constructional aid of Mr. B. Freund, Freund Laboratories.)

Since the tensile behavior of a hair fiber is quite reproducible, particularly if regions of only moderate elongation are studied, a method is available of studying the effect of various chemical, or physical treatments on hair. This is frequently done by comparing the stress-strain characteristics of a fiber, after treatment, with

those exhibited by the same fiber before the treatment. The work required to produce a given elongation of the fiber (proportional to the area under the stress strain curve) is a property that is frequently used for this purpose.

As an illustration of various features of the stress strain curve, the following data are given for hair immersed in water and stretched at a constant rate of elongation of 80 per cent per minute.

Young's Modulus 10^{10} dynes/cm ²	15.25
Yield Intercept, load, 10^5 Gm/cm ²	35.42
Yield Intercept, stretch, %	20.30
Stiffening Intercept, load 10^5 Gm/cm ²	45.50
Stiffening Intercept, stretch, %	24.5260
Ultimate Strength, 10^5 Gm/cm ²	150.250
Ultimate Elongation, %	550.750



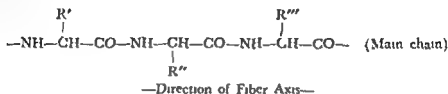
Fig. 64—Extensometer of electric strain gauge type used for study of tensile properties of hair (Courtesy Mr. R. Reed and the Toni Company.)

It must be noted that the actual ranges within which such data may fall are not known accurately. The data given above reflect the results obtained on various supposedly untreated hair samples and are, of course, subject to revision. (Brunner, M. Personal communication.)

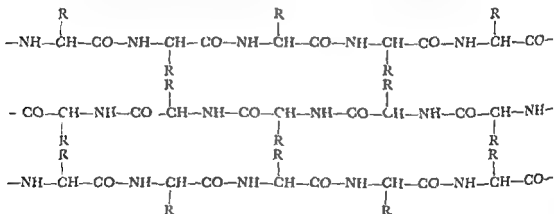
But measurements of elasticity per se are entirely without value unless we recognize the molecular structure of keratin which is the substance from which the hair is built. Astbury's fundamental studies by means of x-ray examinations show

that the interior of a hair is composed of long chain molecules lying within the body of the fiber as fibers lie in the body of a yarn. They are grouped into irregularly defined bundles, running parallel to the fiber. Having many of the same properties as yarn, we find that splitting normally occurs lengthwise, and when distended by soaking in fluid, the swelling measures more across than in length.

The molecular chains which form the basis of keratin are known as "polypeptide chains," the common denominator of all proteins which may be represented in this formula



R' R'' and R''' stand for various univalent radicals such as —H, —CH₃, —CH—CH₃, —COOH, etc., which function as 'side' or 'linking' chains between the main chains and distinguish one protein from another. Polypeptide 'grids' are formed by the main chains running the length of the fiber being attached to one another by these various side chains as follows



Under x ray these grids in unstretched hair prove to be buckled or kinked in appearance, presenting a regularly folded pattern along the main chain to make room for the various interactions of the linking side chains. The elasticity of such fibers can only be understood by considering them as co polymers of a great many polymerizing units. The presence of these diverse amino acid residues or polymerizing units in the fiber prevents a close association of the peptide chains and causes the kinking of the fiber. The stretching of such a fiber involves the unbuckling of these polypeptide chains and this accounts for the rheologic property known as elasticity.

When stretched these buckled grids become flat, but as soon as the stress is relaxed, they return again to their folded pattern. Obviously the simple act of pulling transforms the short intramolecular chain into a large one.

The α keratin of unstretched hair is composed of regularly folded polypeptide chains, but, when stretched, these original molecules become twice as long, forming the β keratin, which always attempts to return to α , or their norm, when the force is relaxed, both a biologic and industrial asset

Water may play an important role in the elasticity of the polypeptide chains. If it is completely absent, certain parts of the keratin molecules adhere to each other so closely that either stretching or contracting is precluded. When the actively charged water molecules are introduced, however, they attract the side chains and divert their magnetism for each other, making the transformation from the α keratin to the β keratin possible. This is called the process of intramolecular lubrication.

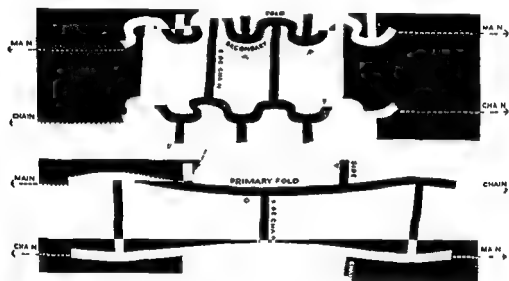


Fig 65—Skeleton models illustrating the essential structural features of α (upper model) and β keratin (lower model) and the change which takes place during the transition from one form to the other when the hair is stretched. Short lengths of two neighboring main-chains only are shown. (After W. T. Astbury, in Savill, *The Hair and Scalp*, Williams & Wilkins Co.)

When the hair reaches its saturation point, retaining an amount of water equal to one third its weight when dry, it may be stretched without damage to one and one half times or even double its normal length. Such a hair, if allowed to dry at this point, will fail to contract to its original length unless it is again thoroughly saturated.

After forcible stretching when the molecule has arrived at its β keratin configuration, stresses are present which in the presence of water, may succeed in breaking the internal linkage of the molecule. When dry the hair is able to withstand the strain if it is controlled within reasonable limits, but when liquids are present, the active molecules of these substances in recombining with the polypep-

side chains complete a breakdown which was initiated, in part, by the mechanical deformation. It is the cross linkages which break under stress, collapsing under the continued action of water, at a rate commensurate with the rise in its temperature or the addition of alkali.

Since these physiologic factors are obviously pertinent to any consideration of permanent waving, discussion is completed in the chapter dealing with this subject (pages 132-141). There it will be noted that after a hydrolytic breakdown of the cross linkages, new, unstressed side chains may be formed in the presence of steam and hot water which will successfully hold the unnaturally elongated molecule in its β keratin state.

Bull and Gutman, in studying the elasticity of keratin fibers, concerned themselves with what they termed the "stress-strain" curves of human hair in pure water. They concluded that while the stress strain behavior of the hair is reproducible up to a 20 per cent stretch, the process is essentially irreversible. They also claim that they established an extent of stretch beyond which one cannot go without causing irreparable damage. This point begins at about an 18 per cent extension.

Furthermore, they experimented to discover the influence of temperature on the elasticity of hair and found that it increases with increasing temperature in the Hooke's law region. The results obtained on other portions of the stress-strain curve were ambiguous except on the contraction limb of the 20 per cent stress-strain loop. In this region, the elasticity decreased with increasing temperature and indicated a behavior reminiscent of rubber.

Because of these findings they concluded that Astbury's explanation of the α keratin given above is too condensed, and they made the countersuggestion that the principal feature of the α -keratin is the close packing of successive alternate amino acid residues along the peptide chain. They also suggest that the stretching of a hair from 3 to 20 per cent involves a process which resembles a thixotropic gel-sol transformation and is mainly responsible for the irreversible nature of the extension-contraction process.

Giroud and Leblond reviewed the x-ray pattern similarities between Malpighian and cornified layers of the epidermis and its derivatives. They observed that Astbury undertook the study of hair with the help of x-ray diffraction techniques. According to them it is well known that a beam of monochromatic x-rays directed onto a crystal will be partially diffracted by the rectilinear planes of that crystal and that such diffracted rays can be focused and recorded on a photographic plate. Such diffracted rays produce a series of spots which can be analyzed for number, location, spread and intensity. Analysis of such data can then be used to make deductions regarding the periodicity of the structures making up the crystal. They mention that Astbury found that human hair and sheep wool did diffract x-rays as if they were crystalline, thus confirming the findings obtained by the birefringence studies. The x-ray pattern indicated that a periodicity of 5.1 μ occurred along the axis of the human hair. Since hair can be stretched 100 per cent in a warm, moist atmosphere without breaking the x-ray pattern was examined under these conditions and a new periodicity of 3.4 μ was discovered. Later other periodicities of higher order were also found. It was Astbury who first suggested that the two

basic periodicities were due to the two forms of keratin, which he called α and β , and concluded that the α type could be transformed into the β type by stretching.

Giroud and Champetier used the horse burr, a soft keratin derivative, to determine whether this material would produce the same pattern as the hard keratin of the hair. These authors separated the cornified from the Malpighian layers, stretched both slightly to line up the presumed inherent crystalline structure, and defatted and dried the material before exposing it to the incident x-rays. They then found that both the cornified and Malpighian layers exhibited a periodicity of 5.1 \AA , identical to the α pattern found by Astbury in the unstretched human hair. They concluded that this α -pattern was common to hard and soft keratin, and pre-existed, as well, in the Malpighian layers, presumably as a precursor of the keratins. Derksen, Heringa, and Weidinger, using the snout of the cow, confirmed the presence of the α -keratin pattern in the Malpighian and soft keratinized layers, and showed that with 100 per cent stretching in warm moist conditions, a β -pattern could be obtained with these structures as with the hair. Mercer examined the x-ray diffraction pattern in the Malpighian layer, keratogenous zone, and cortex of the hair. The α pattern appeared in the upper region of the Malpighian layer and persisted unchanged throughout the keratogenous zone into the hair itself. From all these results, it may be concluded that a protein with the keratin pattern pre-exists in the Malpighian zone and is actually the precursor of the true keratin formed at the limit between Malpighian and keratinized layers.

This concept was confirmed by Rudall who, using a strong solution of urea, was able to isolate from the Malpighian layer of the epidermis a protein which, under proper conditions, diffracted x rays in both an α - and a β -pattern. Rudall's protein displayed long-range elasticity when in fibrillary form and, in most of its properties, behaved as might be expected of a keratin deficient in cross linkages. The extractability of this protein may be noted in parallel with the fact that the tonofibrils of the Malpighian layers are easily modified by enzymes, acids, and autolytic processes, as shown by Behn, Weidenreich, and Patzelt. In contrast, the birefringence (double refraction) of keratinized structures is not easily altered by these chemical treatments, indicating that tonofibrils become resistant following keratinization. Thus, Mercer showed that similar chemical treatments of longitudinal sections of hair remove the birefringence only in Malpighian layers at the level where tonofibrils begin to form. This work included an examination of the x ray pattern, from which it appears that the x ray pattern was also disorganized in the same region by the chemical treatments. Apparently, the keratinization process stabilizes both the birefringence (of tonofibrils) and the molecular pattern.

Garn recently showed that hair like nerve fibers, muscle fibers, and many natural textile fibers, is birefringent, that is, double-refracting. The flattened cornified cells that make up the hair are arranged parallel to the long axis of the hair. Further, the crystallites of keratin possess a similar orderly arrangement, with at least one axis parallel to the long axis of the hair. Thus, the essential birefringence of the component material is preserved by the alignment of the structural units. The hair shaft is birefringent at right angles to the long axis.

Since birefringence increases with the degree of keratinization the cortical cells are more birefringent than the medullary cells and the more flattened cells nearer

the neck of the follicle are more birefringent than the cells nearer the center of proliferation. Thus, the optical properties of the hair can be correlated with data derived from chemical analysis, differential staining, x-ray diffraction spectra and crystallographic studies.

Hamburger has also performed some interesting experiments relevant to the elasticity and mechanical behavior of hair. Due to the variation of hair types (the variations within a class often being greater than the variation between classes) a classification becomes exceedingly difficult, and the task of obtaining data of significance would therefore necessitate an enormous number of samples. He devised a parameter for characterization, consisting of the ratio of immediate elastic deformation to total deformation. Elasticity is measured by the rate of travel of sound down the hair shaft in micro-seconds. Correlating data on this parameter, it has been found that a classification of good agreement is found to group "problem hair" and children's hair on the one hand, as against normal hair on the other. The normal hair shows consistently greater elasticity than the first group. Chemical treatment (bleaching thioglycolates) reduces, but normal sunlight bleaching increases, elasticity, it was reported. Oxidation and low temperatures increase, reduction decreases elasticity. Wetting lowers yield point, but does not effect elasticity below this point. Since the stress strain curve has three regions, immediate elastic deflection, delayed deformation (recoverable and permanent), mechanical measurements are best made on the early section at low strain (4 to 5 per cent extension), the hair pulls out of the head at the end of this region at about 45 per cent of the rupture load. Hamburger stated that hair being a high polymer, it does not follow the laws of elastic behavior. The stress strain curve of human hair is apparently intermediate between those of acetate and wool.

PERCUTANEOUS ABSORPTION AND PERMEABILITY

Many factors complicate the scientific study of percutaneous absorption. The skin itself varies in thickness in differing parts of the body, ranging from 0.01 to 0.02 mm. in the case of facial skin from the cheek, to 0.60 mm. on the palmar and plantar surfaces. There is no way in which one can compute an 'average thickness'. Then too human skin is unique because the sweat glands generally open in the surface independently of the hair follicles due perhaps to the atrophy of our prehistoric furry coat while other mammals have sweat glands which open into the follicles above the sebaceous gland ducts. Because of this basic physiologic difference it is not always possible to assume conclusions for human beings from experiments which have been carried out on animals. The passages from the outer skin include not only sweat and sebaceous gland channels, but the hair follicles which open on the surface in the form of visible ostia. Since the hair shaft does not adhere to the follicular wall, there is a space between the two which is loosely filled with greasy, horny scales and air—a continuation of the duct of the sebaceous gland which empties its sebum into it. Any substance may reverse this process and reach the duct of
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transitory epidermal cells. In the same way, substances may find entrance to the sweat glands through their ducts.

In spite of these orifices which appear to aid absorption in man, it seems that the property of the two-way passage such as the frog's, has become restricted so that, while retaining its excretory functions, the skin may have lost not only most of its passive (permeability) but also its active (absorptivity) capacity of the transference of substances inward. Until 1877, medical men believed that gases and volatile substances penetrated the skin freely. When the investigators started to examine this belief from 1877 to 1900, their experiments led them to state vehemently that the skin was absolutely impermeable to all substances. From 1900 to the present, research experts using more refined analytical techniques, determined that many substances are more or less able to pass through the skin. The consensus may still be characterized as cautious.

Attempts have been made to study the permeability of the skin and scalp to fats by the use of histologic and histochemical methods. While these techniques have shown the probable avenues of entrance to be the apertures of the hair shaft and the glandular ducts, they have given no completely satisfactory explanation of absorbed fat below the rete malpighii and in the cutis. The only visible penetration of fats is in the appendages. In fact, oils, fats, and aqueous substances in general do not penetrate the intact epidermis to any appreciable extent beyond a limited surface diffusion reaching a maximum in lard and vitamin F, the active isomer of linoleic acid, but in no case did this amount penetrate to more than one third the depth of the stratum corneum. On the thinnest scalp surfaces, such slight penetration may possibly exert a temporary lubricating influence on dry, flaking skin. The success of oleic acid is perhaps attributable to the hydrophilic group at one end of the molecule. Mineral, vegetable, and animal fats are permeable in various degrees according to the ascending order (Harry).

Essential oils such as clove, eucalyptus, and turpentine, and that of oleic acid have comparatively excellent penetrating properties. Cod-liver oil, lanolin, lanolin absorption bases, lards, goose grease, so-called vitamin F and oleyl alcohol may be judged as good, along with the following oils: arachis, avocado pear, grape-seed, olive, castor, and turtle, as well as hydrogenated arachis whose absorption was not inhibited by hydrogenation. Liquid paraffin and petroleum jelly proved to have negligible penetrative powers.

Harry notes, as does Lang, that the presence of polar groups either with or without water was found to confer penetrant properties upon mineral oils and greases otherwise impermeable and to improve their adherence along the skin surface, but no evidence was obtained to confirm the theory that the molecular chain length of the fat molecule was of major importance in skin penetration.

Calvery, summing up the experiments of many investigators, concluded that mercury and heavy metals were permeable through pilosebaceous channels and could be checked by urinalysis. Arsenic, lead, and copper oleate in a lanolin petrolatum base penetrate the skin. Mercury passes through the skin (including the scalp) in small quantities and can be found in tissues, body fluids, and excreta by routine chemical analysis. *Proteins in minute quantities may penetrate the skin*

and circulate in the blood under certain conditions and insulin also passes through the skin from ointment bases containing Eucerin or Lanolin or even alone. Amino acids are resistant to being absorbed since they may be considered lipid insoluble. There is a single report claiming the penetration of cystine through the skin with its subsequent detection as a rise in sulfur in the urine but until further studies are reported amino acids remain among the substances which the skin does not absorb. Up to date there are too many conflicting reports about carbohydrates to report any conclusive evidence of their permeability. There is however, ample evidence that hormones and vitamins A B C D and K pass through the skin. The problem of vitamin and hormonal passage raises the question however as to whether the amounts absorbed are actually adequate for percutaneous therapy.

In spite of the fact that penetration does occur following the use of local applications the skin cannot receive its basic nutritive materials by the small percentage so absorbed. As has already been stated the skin derives its major source of nutrition from its blood supply.

HAIR EXCHANGE AND HAIR GROWTH

Hairs age. There is an unbroken and unobserved cycle of growth fall and replacement of hairs on the human body contrasted with the periodic exchange of animal hair. When they reach the end of their life span they are shed having become loosened from the papilla by the formation of an air space between its top and the medulla which causes the disappearance of the cuticle at this level. Then the root sheath grips the shaft and slowly moves the hair to the surface. As it leaves the papilla the hair sends down a column of cells which becomes the nucleus for the growth of a new hair in the old follicle gradually displacing its predecessor which in its final stage has become only loosely attached to the walls of the follicle and is easily removed by combing brushing or effortless traction.

If the old hair has not left behind a vigorous offshoot column of cells no new hair will develop in the old follicle. On the other hand if the required strand of cells has been left in the follicle it takes a brief time for the new hair to develop from this germ assume its structural characteristics and assert the pressure necessary to help push out the old hair and eventually appear on the cutaneous surface. The regeneration of the papilla is supposedly due to a renewed supply of blood for its nourishment from the vascular plexus which feeds the base of the follicle.

An investigator interested in observing the production of a single follicle for six years carefully watched a terminal hair which was growing from a small nevus on the back of his hand. During all these years this single follicle was responsible for the production of fourteen different hair shafts. One of these was present at the beginning of the observational period and one was still living when his findings were published so actually the complete lives of only twelve separate shafts were checked from their first appearance to their eventual shedding. It is interesting to note that in these six years there were only about twenty four days during which no hair at all was visible on the cutaneous surface. Even though all the hairs observed had their genesis in an identical matrix each proved to be distinctive no two appearing to have a completely similar structure. Highly individual characteristics were noted in each replacement.

While rats have a total cycle of hair growth of thirty five days from the formation of the hair bulb to its separation from the papilla the human hair lives an average of one hundred and eighty days. It is found that summer hair regularly displays a superior longevity maintaining an average of one hundred and ninety seven days before shedding.

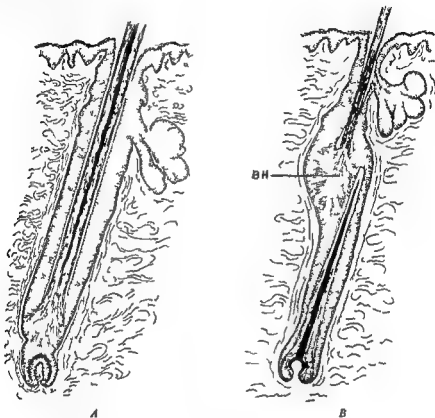


Fig 66—A Early stage of the shedding of a hair showing its separation from the papilla. B Later stage of hair shedding showing a bed hair (BH) and a new hair growing from the same papilla. (Courtesy McCarthy Diseases of the Hair)

Hair growth is intimately associated with the function or dysfunction of certain glands of internal secretion and the major aspects of this problem are discussed in the section on endocrine physiology of the scalp (pages 97-109). In brief, there seem actually to be three main factors concerned with the growth and development of hair. The first of these may be called a constitutional factor representing the capacity of an individual hair follicle to respond to endocrine stimulation. The second is a hormonal factor supplying the stimulus for hair growth and to those follicles particularly sensitive to such action, and the third may be summed up as local influences involving blood supply, autonomic innervation, and systemic nutritive factors affecting the hair-producing follicle.

Ambosexual hair remains normal even after prepuberal castration. Since androgen therapy increases both the size and number of pubic sebaceous glands in

III Pituitary (anterior lobe)

- a Gonadotropic hormones (follicle stimulating, luteinizing)
- b Growth hormone
- c Thyrotropic hormone
- d Adrenocorticotropic hormone

IV Thyroid

Since the most widespread and obvious effects on the pilosebaceous system originate in the endocrines concerned with sexual development, the male and female gonads exerting androgens and estrogens, respectively, and the adrenal cortex which produces both in both sexes primarily absorb our attention

I Gonads

Estrogen is one of the ovarian hormones. It stimulates the development of the muscular, epithelial, vascular, and secretory components of the genital tract. The administration of estrogens results in certain effects on the pilosebaceous system namely, a diminished activity of the sebaceous glands, a shrinking of the follicular orifices and the diminution of keratinization. As a result, estrogen therapy is often followed by the appearance of smooth, delicate skin, drier and fluffier hair, the diminution of dandruff, softening of the beard, and, in patients with acne, the involution of comedones and the subsidence or disappearance of pustules (acnei form). Although the statement is frequently encountered that estrogens also increase or stimulate the growth of scalp hair, there is no indubitably conclusive evidence of such an effect.

Estrogenic substances occur widely in nature, in plants as well as in animals. Included among the natural estrogens are estradiol (dihydroxyestrin), estrone (ketohydroxyestrin), and estriol (trihydroxyestrin) which are extracted from urine or placentas of human beings during pregnancy, while several estrogens including estrone, equilin and hippulin are obtained from the urine of pregnant mares. Sow ovaries contain both estrone and estradiol but not in sufficient quantities to make them a commercially worthwhile source. Estradiol exists in two stereoisometric forms—alpha and beta. The alpha estradiol is probably the most potent of all known naturally occurring estrogens; the beta form is relatively inert.

Dodds and his co-workers, after extensive experimentation with synthetic substances, discovered the estrogenic activity of the stilbene compounds. Diethylstilbestrol is one of the most potent of these compounds yet synthesized. It may be prepared in a variety of ways from nonbiologic organic chemicals. Its physiologic activity duplicates practically all the known actions of natural estrogens and it also inhibits the secretion of various factors of the anterior lobe of the pituitary gland. It differs in its action from natural estrogens in its inability to cause the ovipositor reaction of the female bitterling and to antagonize the action of androgens on comb growth of capons. Various modifications of diethylstilbestrol have been devised such as fatty acid esters and a number of ethers for increasing the estrogenic efficiency of this substance. Diethylstilbestrol possesses the advantage of being highly active by mouth as well as percutaneously. The ratio of potency between oral and parenteral administration varies in the hands of different investigators from 1:2 to 1:5 in the human being as well as in rodents.

Ethinyl estradiol is a variant of estradiol which does not occur biologically and is the most potent, orally effective estrogen. While this compound does not occur naturally, it reproduces all the physiologic and therapeutic effects of estradiol.

Androgens are derived from the male gonad and from the adrenal cortex. Their function is to promote the growth and development of the genital organs, an active libido and male secondary sex characteristics. They stimulate the surface epithelium of the skin by their mitotic action and accelerate the production of sebum. They are also responsible for the hyperkeratosis of the follicular orifices. Androgens are of primary concern in the problem of male pattern baldness.

Testosterone has been isolated from testicular tissue and has been synthesized. It is secreted by the interstitial cells under the influence of the anterior hypophysis and is responsible for the development and maintenance of the accessory male organs and secondary sex characteristics. It also has important physiologic functions in protein anabolism. Following castration in the male, the seminal vesicles, prostate, and penis undergo atrophy, libido and sexual activity are diminished. Parenteral and oral administration of testosterone and companion preparations restores these structures and functions to normal, but beneficial effects in castrates or eunuchoids are present only as long as replacement therapy is continued.

In the male, testosterone therapy is primarily employed to supply testicular hormone for the treatment of deficiency or absence of this internal secretion of the male. It is therefore of value as replacement therapy in the treatment of prepubertal and postpubertal eunuchism or hypogonadism (deficiency states) and in postcastration or eunuchism from other causes. Its use in eunuchism is intended to promote the development of primary and secondary sexual characteristics of patients with organic testicular failure, after the age of 16 or 17 when puberty has not occurred spontaneously, and to relieve postpubertal constitutional symptoms attributable to deficient secretion.

The most interesting clinical feature of the gonads is that while androgens encourage the growth of coarse body hair along male configurations, the estrogens inhibit its growth, whatever hair remaining being female in its distribution. However, the exact opposite effect is produced in the hair follicles of the temporofrontal and vertex regions of the scalp, where, according to Barber, growth is encouraged by estrogens alone. He states that in female cases of male pattern alopecia, estrogens will stimulate the growth of hair on the scalp even when applied locally. He even makes the observation that in advanced cases considerable regrowth of hair may be achieved. Thinning of the scalp hair is a not uncommon component of the climacteric period. In many women of the menopausal age, observed by me and by others, large amounts of estrogen administered both locally and parenterally failed to initiate a regrowth of hair although it often stopped further hair fall. In similar vein, the therapeutic use of estrogens either to control or to improve ordinary male baldness, lacks both statistical proof and controlled observations. We can only state that an excessive production of androgens combined with hereditary and racial factors (plus various unknowns) act as the precursors of ordinary baldness.

Men castrated before puberty, and men whose gonads have remained infantile with consequent psychic and physiologic characteristics of the eunuch, known as eunuchoids, both have scanty body hair of female distribution, sparse beards or none at all, but thick, luxurious hair of the scalp. Such persons do not become bald. Hamilton carefully observed 104 eunuchs and eunuchoids. He proved that appropriate doses of androgens matured the sexual apparatus of those with delayed glandular development, stimulated the growth of coarse, male pattern body hair, and at the same time, precipitated male type alopecia in those with an hereditary predisposition (see page 173). Others who merely have a relatively low production of androgens fall into the class of men popularly described as the Casper Milquetoasts of life. They are often physically weak, complaining, depressed, sluggish in thought and action, and therefore often economic failures. One worry alone does not concern them—they are unlikely to suffer from ordinary baldness. This observation should not be interpreted as evidence in support of the theory that hairy-chested, bald men are more virile than others.

Rony and Zakin investigated some of the effects of endocrine substances on the adult human scalp. Their first subject was an 18 year old boy with beginning male pattern alopecia; their second, a man of 34 with an extensive alopecia areata which in three years had denuded 90 per cent of his scalp. For the younger man 40 mg of methyltestosterone by mouth was prescribed daily for the first three months. Then treatment was suspended for a month but was followed by 5 mg of diethylstilbestrol by mouth for another three months. There was an increase in body hair and his overdry scalp hair became oily, but no regrowth was observed. Histologic sections showed a decided increase in the number and size of the sebaceous glands on the scalp. During the ingestion of diethylstilbestrol, his mammary glands became painfully enlarged, hyperpigmented and sensitive. The older patient who, like the first, had a normal basal metabolic rate, was given 40 mg of methyltestosterone orally, plus 25 mg of testosterone propionate twice a week intramuscularly for four months. During the following month he took 5 mg of diethylstilbestrol daily by mouth. He suffered from pruritus of the scalp during the period of androgen therapy and the effect was negligible as far as hair growth was concerned. The enlarged sebaceous glands characteristic of alopecia areata were subject to further hyperplasia by the end of the five month interval, and histologic sections showed a definite atrophic effect on these secretory glands. During the period of estrogen therapy, hair fall increased.

The authors concluded that androgens have a stimulating effect on the sebaceous glands of the scalp regardless of location or age of the subject. The effect was even powerful enough to increase the already hyperplastic glands in a patient with alopecia areata. On the other hand estrogens caused a reduction in the size and number of the sebaceous glands in human beings as Hooker and Pfeiffer demonstrated in rats. The important observation was that since androgens do not promote the growth of scalp hair in cases of premature baldness, the response provoked by androgen in the hair follicle must be a distinct and different phenomenon from the response provoked in the sebaceous glands. One depends on factors of a regional character inherent in the responding tissue while the other seems to be independent of such controls.

In women, androgens, whether derived from the adrenal cortex or from an androgen producing ovarian tumor (arrhenoblastoma) have the same effect when they unbalance the all important ratio between androgen and estrogen production. Male pattern alopecia in women may occur as a result of androgen preponderance. This imbalance is not due to estrogen deficiency alone but rather to a disproportionate amount of androgen production. This hypothesis is suggested by the fact that hirsutism can be induced in women by the administration of sufficient dosage of androgens whereas hirsutism of itself is invariably associated with estrogen deficiency states. The problem is further complicated by 17 ketosteroid excretion studies in women with hirsutism which revealed no significant correlation between androgen excretion and the presence of hirsutism. The hirsutism may be the result of some intermediate product of the steroid metabolism which is not detectable by our present methods. Some degree of masculinization may be noted in the pilosebaceous system when the ovarian adrenal relationship has been disturbed by physical and psychic stresses. Other changes may be noted during pregnancy and menopause. It is a known fact that the estrogen content of the blood continuously increases from the end of the second month of pregnancy until term reaching a level which is much higher than that of premenstruation. Women with the seboreic diatheses and even male pattern alopecia, at the end of the second month of gestation observe that their scalps are less oily, the sebum diminishes to a normal amount and the amount of hair fall is temporarily arrested. Scalp hair, and skin components (acne) usually share in this improvement. Barber and others attribute this improvement to the rising estrogen levels of pregnancy, an attractive but unproved theory. Of related interest is the fact that several weeks after childbirth hair fall occasionally occurs as after a toxic or febrile disturbance. The observation has been made that the patients who develop diffuse alopecia following childbirth manifest an extreme degree of estrogen deficiency as determined by vaginal smear studies. This phenomenon is possibly attributable to an inhibiting effect on the gonadotropic activities of the anterior pituitary resulting from the high steroid hormone levels occurring during pregnancy. The estrogen deficiency state is associated with other symptomatic features of estrogen deficiency namely vasomotor symptoms (flushes) and amenorrhea. Normally the estrogen deficiency state is of short duration lasting for several weeks and is spontaneously corrected by the normal resumption of the pituitary ovarian hormonal cycle.

The failure of the restitution of this cycle results in a variable degree of alopecia which may be of permanent duration but which usually is followed by regrowth of the scalp hair. It is worthy of note that in a few of these patients I have been able to observe a lowered basal metabolic rate which suggests the possibility that the alopecia may be ascribable to a deficiency of more than merely the gonadotropic fraction of the pituitary gland namely the thyrotropic and possibly adrenocorticotrophic factor as well. Unfortunately definitive hormonal studies have not been reported on this subject. This offers a fertile field for further research. In this connection the author in collaboration with Dr U J Salmon was privileged to study a patient with alopecia totalis during her pregnancy.

Prior to pregnancy this patient had a normal menstrual cycle. During pregnancy she developed several patches of hair on her scalp beginning at about the

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Prior to pregnancy this patient had a normal menstrual cycle. During pregnancy she developed several patches of hair on her scalp beginning in the first trimester.

third month. At this point, pregnandiol, estrogen, and 17-ketosteroid urine output was found to be normal for this stage of pregnancy. These determinations were repeated at intervals of six to eight weeks and were found to correspond with the normal values for pregnancy. Gonadotropin values were essentially normal.



Fig. 67—Partial regrowth of scalp hair during the pregnancy of a patient with alopecia totalis.

Corticosteroid determination performed two weeks preceding delivery was found to be 41 mg. The significance of this high value and its possible relationship to the growth of scalp hair are a matter for conjecture inasmuch as this data merely indicates hypersecretion of one of the adrenal cortical hormones. It is significant that within two months after delivery the scalp hair which had grown during pregnancy was completely lost. Why did this woman grow hair during the pregnancy which she lost within two months following the termination of her pregnancy? It is tempting to attribute the growth of hair to the sterol sex hormones, particularly the progesterone which is produced in huge amounts by the placenta. During the last trimester of her pregnancy the daily pregnandiol excretion varied from 60 to 85 mg (maximal daily level during the ordinary menstrual cycle is 10 mg). The therapeutic potentialities of progesterone with respect to hair growth are being investigated.

During the menopause the declining estrogen levels may cause the scalp hair to become thinner and finer in texture. Diffuse hair fall sometimes occurring in the decade between 50 and 60 may be effectively checked by local estrogen injection and by the oral and intramuscular administration of thyroid and estrogen in adequate dosage. If the alopecia is of the male configuration and is present

the primary factor may be an overproduction of androgens by the adrenal cortex rather than an ovarian deficiency. In such cases estrogen therapy will not prove efficacious. In any case, there are varying degrees of tissue sensitivity to the sex hormones since the follicular apparatus of the scalp may differ considerably in its reactions to the gonadal secretions.



Fig. 68—Facial hirsutism as a manifestation of the adrenogenital syndrome. (From Behrman: *Dermatologic Clues to Internal Disease*. Grune & Stratton. Original photograph by Dr. I. C. Rubin.)

Winsauer and Manning studied a patient with a masculinizing tumor of the ovary. Their patient had coarse hair covering the face, trunk and arms with masculine pubic hirsutism. Her scalp hair was thin and fine, with recession at the temporal hairline. Her urinary excretion of 17 ketosteroids in 24 hours was 43 mg, which dropped to 17 mg postoperatively. Koets also checked the 17 ketosteroid excretion levels in nine cases of idiopathic hirsutism, measuring the levels at weekly intervals during complete menstrual cycles. There was a cyclic variation with maximum excretion at the approximate time of ovulation in contrast to the relative

constancy of excretion in normal women, and reaching a level well above the normal limits. The authors advanced the theory that hirsutism is probably connected with hypersensitivity of the adrenal cortex to ovarian hormones.

II Adrenal Cortex

The effects of the adrenal and pituitary glands on hair growth cannot be specifically delineated because the precise interrelationships of the adrenals, the pituitary, and the various tissues of the body are not clearly defined. Moreover, the functions and modes of interplay of the individual steroid hormones produced by the adrenal cortex are extremely complex. In many respects, therefore, a full understanding of how these factors operate to influence hair growth and various physiologic and pathologic states remains to be attained.

The present concept is that the secretion of cortisone and other cortical steroid hormones is mainly or entirely mediated through stimulation of the adrenal cortex by the adrenocorticotrophic hormone, this hormone is elaborated by the anterior pituitary. The function of the anterior pituitary is presumably controlled to some degree by the hypothalamus, which, in turn, can be influenced by higher brain centers. The adrenocorticotrophic hormone has the specific function of regulating the activity of the adrenal cortex. It is in response to the action of this hormone that the adrenal cortex secretes its steroid hormones.

The direct or indirect humoral pathway between the hypothalamus and anterior pituitary is only one of the ways in which the gland is stimulated to produce adrenocorticotrophic hormone. Another regulatory mechanism involves the adrenal medulla which, in response to stress, releases epinephrine. It is now believed that epinephrine, reaching the anterior pituitary via the systemic circulation, causes a release of adrenocorticotrophic hormone. However, this effect has thus far not been found of practical value from the standpoint of therapeutics.

The cortex of the adrenal gland is essential for life. Adrenalectomized animals die in a few days. Effects of acute adrenal insufficiency, in disease or after experimental procedures in animals include blood concentration, low blood pressure, gastrointestinal disturbances, asthenia, subnormal temperature, and low basal metabolic rate. There also may be loss of sodium and retention of potassium in most species, loss of carbohydrate reserves with hypoglycemia and retention of nitrogenous products in the blood.

Extracts of the adrenal cortex contain several potent substances which influence electrolyte, water, or carbohydrate metabolism to various degrees, however, as demonstrated on small animals, no one of these substances and no synthetic substance possess all the effects of a potent cortical extract. There have been isolated from the cortex crystalline compounds which are capable of maintaining the life of adrenalectomized animals and restoring toward normal the disturbed metabolic conditions induced by adrenal insufficiency. These compounds are steroids. The most potent are desoxycorticosterone, corticosterone, dehydrocorticosterone, and 11 dehydro 17 hydroxycorticosterone. Many other steroids have been isolated from this tissue but most of them have little known physiologic activity and probably but slight influence on the growth of scalp hair.

Cortisone and ACTH are complex hormonal substances capable of affecting many body functions and many tissues. Metabolically, they affect electrolyte balance by producing sodium and water retention, hypopotassemia, hyperglycemia, and a negative nitrogen balance. Both cortisone and ACTH also retard wound healing and produce moderate elevations of the blood pressure. In some patients it is apparent that these drugs effect excessive adrenal cortical changes such as rounding of the face, mild hirsutism, acne, striae of the skin, and, in a few instances, seborrheic dermatitis. The hirsutism is similar to that described in patients showing effects of excessive production of adrenal cortical hormones. In the occasional case there is also a moderate accentuation of loss of hair from the scalp if a tendency toward alopecia of the male type has previously existed. These effects may also be due to an increased protein catabolism or to the effects of these drugs on the basal metabolic rate. As is known, both hormones either may depress thyroid activity, probably via diminished pituitary production of thyrotropic hormone, or they may increase the sensitivity of the tissues peripherally to thyroid hormone.

The chemical structure of the cortical steroids is closely related to that of the sex hormones, in fact, some of the cortical steroids have estrogenic or androgenic properties and in certain abnormal conditions of the cortex, large amounts of estrogens or androgens may be recovered in the urine. On the other hand, the sex hormone progesterone has life maintaining properties in adrenal insufficiency in small animals, while other sex hormones such as estrone and testosterone are capable of inducing slight electrolyte changes similar to those produced by cortical steroids.

While our knowledge of the identity and significance of the 17 ketosteroids has advanced considerably in recent years, we still know very little regarding the nature of the corticosteroids in urine. So far, only one corticosteroid, 17 hydroxycorticosterone, has been identified in urine. Urinary corticoids have been referred to in the literature by various names such as cortin like material, glucocorticoids, and 11 oxysteroids.

The adrenogenital syndrome is directly caused by an overproduction of adrenal androgenic hormones and, in contrast to Cushing's syndrome, is usually without metabolic disturbance. It has been found associated with adrenal hyperplasia and with adrenal neoplasm. However, it may also occur without any demonstrable changes in the adrenal glands, and in this event may either be on a purely functional basis or, rarely, may be due to pathology in undetected adrenal rests. A similar clinical picture is also produced by certain ovarian tumors.

The adrenogenital syndrome may be subdivided according to Wilhelm by means of the sex and the stage of development at which cortical hyperfunction begins.

- 1 Embryonal (a) Female : pseudohermaphroditism (b) Male : macrogenitosomia precox
- 2 Postnatal (a) Female : virilization (b) Male : feminization

The direct causal relation between female pseudohermaphroditism and adrenocortical hyperfunction is now generally accepted, according to Wilhelm. The adrenal hyperfunction in these cases begins in utero, the female embryo being profoundly masculinized with enlargement of the clitoris, malformation of the external

genitalia and hypertrichosis. These patients are often compelled to shave daily and have a tendency to male pattern baldness. The body form is masculine in type, short and usually muscular. Female pseudohermaphroditism is almost always due to bilateral adrenal hyperplasia, although rarely unilateral hyperplasia or tumor has been found. The urinary 17 ketosteroid excretion is considerably elevated and the enlarged adrenal shadows can be clearly delineated on the laminographic films following perirenal insufflation.

Adrenocortical hyperfunction occurring in males during infancy and childhood is manifested by precocious sexual development, rapid growth and aging. The skeletal and muscular systems are so far advanced that these boys appear older than their actual age—the infant Hercules type. Facial and body hair appear, with deepening of the voice, and sometimes obesity. The penis resembles that of an adult.

When adrenocortical hyperfunction begins in childhood or adulthood the masculinizing effect in females is less profound than when it begins in embryo. The clitoris is but slightly, if at all, enlarged, and the external genitalia appear normal. When virilization begins before puberty these patients have a masculine body form, absence of breast development, a low pitched voice, considerable increase in body and facial hair, and amenorrhea.

When on the other hand adrenal hyperfunction begins after puberty the already developed breasts become smaller and flaccid, but some evidence of the female contour persists. Menstruation is scanty or suppressed. Obesity is not uncommon.

Virilism may be associated with adrenal tumors, with adrenal hyperplasia and also with certain pituitary and ovarian tumors. On the other hand virilism frequently occurs without any demonstrable pathologic changes either in the pituitary ovaries or in the adrenal glands. These women are usually young, often obese, and their chief complaints are related to increased facial and body hair. Thorough investigation of these patients fails in most cases to reveal definite evidence of any gross endocrine lesions. In a minority, however, the urinary 17 ketosteroids were elevated, suggesting adrenal hyperfunction. Salmon has described this group of patients as the arrhenomimetic syndrome. The paradoxical feature of some of these cases is that while the hypertrichosis is masculine in pattern (in some instances being so marked as to require daily shaving) this does not necessarily interfere with the normal reproductive physiology. Several of these women have become pregnant and have given birth to normal children.

III Pituitary Gland

The effects of the pituitary gland on the growth of scalp hair are primarily due to the action of components of its anterior lobe.

The pituitary gland, especially its anterior lobe, secretes hormones which are one of the major correlatives of the endocrine system. It is these hormones which stimulate the activity of the adrenal cortex. The latter would waste away should its pituitary nourishment be denied. Failure of the adrenal cortex to function might be due either to the exhaustion of the adrenal itself or to the diminishing power of the adrenotropic action of the anterior lobe of the pituitary.

A number of factors are concerned in the action of extracts of the anterior lobe (1) a growth factor which influences the development of the body, (2) a factor which stimulates the growth and maturation of the ovarian follicle, which in turn bring on the changes characteristic of estrus (3) a factor which causes luteinization of the ovarian follicles, (4) a factor which is necessary for normal thyroid development and function and which, if present in excess, produces hyperplasia of the thyroid with hyperthyroidism in both the rat and the guinea pig. In addition to the above factors, whose existence is established, experimental evidence indicates the presence of other principles. Among these is adrenocorticotrophic hormone (ACTH) which stimulates the adrenal cortex.

Hypersecretion of various anterior pituitary fractions results in acromegaly and is characterized by the magnification of various parts of the body. The pituitary itself becomes enlarged and develops an eosinophil adenoma of its anterior lobe. There follows a gradual enlargement of the hands and feet, the bones, the cutaneous tissue, the tongue and lips. The size of the hair papillae increases, the follicular orifices are amplified while the sebaceous and sudoriferous glands become hypertrophied. The hair of the body in both sexes becomes coarse, wiry, greasy, and thick. At the same time, patchy alopecia involves the scalp, sparing the lateral and posterior margins. The fingernails become thick and brittle, and the skin becomes free of its substructure and may be lifted in folds between the thumb and forefinger. When the scalp itself is thrown into thick folds, resembling the furrows of a plowed field, it is technically known as *cutis verticis gyrata*, but in popular parlance bulldog scalp.

Cushing's syndrome is due to lesions involving the pituitary gland, often a basophil adenoma of its anterior lobes, with secondary involvement of the adrenal cortex. A similar picture may also be produced by primary lesions of the adrenal cortex. The problem of etiology is less confusing when it is realized that Cushing's syndrome is undoubtedly an expression of increased elaboration and secretion of adrenal steroids. The excretion rate of 17 ketosteroids goes hand in hand with the degree of masculinization. This disease affects women primarily and is characterized by hirsutism of the face and body, accompanied by a thinning of the scalp hair particularly over the vertex and temporo-frontal regions. The skin assumes a dusky color, and red brown, or purple *striae distensae* appear on the abdomen. The skin also becomes dry and scaly, bruising easily. From the ankles to the knees on the anterior aspect of the legs and skin is thin, shiny and subject to diffuse brownish or bronze pigmentation. Other features of the syndrome include hypertension, rounding of the face, buffalo obesity, osteoporosis, acne, hyperglycemia and increased excretion of C_{11} steroids in the urine.

Simmonds' disease is a condition caused by hypopituitarism with the involvement and consequent dysfunction of the thyroid, adrenal and sex glands. The scalp hair dulls and becomes dry, scanty and brittle. The outer half of the eye brows falls out and the pubic and axillary growths are completely lost. In these cases, as well as in pituitary deficiency dwarfs in whom both the adrenal cortices and ovaries are atrophied, panhypopituitarism and pituitary cachexia, Addison's disease and hypothyroidism, the excretion of 17 ketosteroids is extremely low or absent.

Hypopituitarism in human beings acts very much as it does in dogs. When the anterior lobes were removed from young dogs the hair became stiffer, exceedingly dry, and tended to fall out in patches. In mice and young dogs fed the whole gland or injected with the growth hormone secreted by the anterior lobe, the fur grew smooth, thick, and glossy and remained this way even at an age when shagreen is normally to be expected.

IV Thyroid

The thyroid gland regulates the basal metabolic rate and therefore affects body growth. Hyperfunction results in Graves' disease or exophthalmic goiter. The skin is thin and smooth to the touch, the palms of the hands are warm and moist, in contrast to the damp, cold handclasp of the neurotic. There is a diffuse pigmentation, particularly of the eyelids and abdomen, and where thyroid heart disease has developed, the skin becomes salmon pink. The scalp hair is usually abundant but fine in quality, given to premature graying. However, McCarthy calls attention to a study of 111 cases of exophthalmic goiter, all of whom suffered from either partial or extensive alopecia. Sabouraud went so far as to state that some cases of alopecia were directly due to hyperthyroidism. Although there is some factual support of this observation, the reverse is far more frequently the case.

Thyroid deficiency may begin early in fetal life and result in cretinism. The disease marks its victims with a flat bridged, broad nose with wide nostrils, thick lips held far open for a protruding tongue, a dry puffiness of the face, spadelike hands, supraclavicular pads of fat, and a bulging abdomen. Dwarfism and idiocy are concomitant features. The scalp of the cretin is abnormally dry, and hair growth is scanty with a tendency to loosen at the slightest traction. Basinger could produce cretinism with its attendant changes in the pilosebaceous system in more than 50 per cent of thyroidectomized rabbits. Their hair became very dry and was easily pulled out. Only a limited number regained normal coats even with thyroid therapy.

A subnormal function of the thyroid gland acquired after birth results in myxedema with its thickening of the skin, its blunting of the senses and intellect, its labored speech due to an edema of the tongue and brittle, striated nails. The skin becomes pale, coarse, and so dry and scaly that it frequently exfoliates as a fine powder. Areas of yellowish brown pigmentation may be found on the face, neck and forearms. The scalp hair becomes coarse, dry, and lusterless, and falls out until there is only a scanty amount covering the scalp. The pubic and axillary growths may disappear entirely. Hair fall may occur as a ribbonlike band at the forehead and back of the neck. Hypothyroidism is often associated with alopecia areata, ophiasis, or frontal band alopecia. Histologic studies of myxedema reveal atrophic changes in the thyroid glands. The skin shows a relative hyperkeratosis with some keratotic plugging of the hair follicles and sweat ducts. There is also some degree of thickening and obliterative change in the smaller blood vessels.

V Parathyroid

Deficiency in the hormone production of the parathyroid gland leads to tetany, a disease caused by hypocalcemia with increasing neuromuscular irritability and

eventual spasm. Chronic or latent tetany may be associated with ectodermal trophic changes which include atrophy of the nails and a diffuse thinning of the hair. Total alopecia may occur during acute exacerbations of the chronic type.

NUTRITION

At the present stage of our knowledge, it can only be said that diet exerts a definite influence on the scalp and hair. What specific components in the diet are of greatest importance is still a matter for speculation and future research.

Carbohydrates in excess are contraindicated in many infectious disorders of the scalp as well as in the seborrheic state. Specific changes in the scalp due to excess or lack of carbohydrates are unknown.

Fats play a role in the proper function of the sebaceous apparatus. In this respect, the experimental work of Pollicard and Tritchovitch is enlightening. These authors showed that scarlet red, when given by mouth together with food, produces vital staining of the depot fat in the subcutaneous tissues and omentum, the composition of this stored fat depending very largely on that of the lipids ingested. It was observed, however, that the tissue fat disappears, and with it the coloring dye. These authors demonstrated, moreover, that the cells of the sebaceous glands normally elaborate their fatty secretion from the fatty acids circulating in the blood. This is a process of true cellular activity. However, these cells are also capable of taking up the lipid particles that circulate in the blood, especially after digestion. These particles, unlike the normal sebaceous secretion, are colored after ingestion of scarlet red. They are found extending to the periphery of the gland, in close contact with the capillaries, the normal sebaceous fat, in contrast, is localized in the center of the gland, near the duct. Thus, the normal function of the sebaceous glands is adipogenesis, the manufactured fat having a special composition of its own. Under certain conditions—for example, in animals after hyperalimentation—the sebaceous glands assume the function of adipopexis (fat fixation), in which case the fat taken up directly from the blood may be of the same character as the ingested fat. These observations make it easy to understand how the normal sebaceous secretion may be altered either by an excessive intake of fat or fat-forming foods, or by ingestion of special forms of fat.

Burr and Burr found that unsaturated fatty acids are essential for the proper nutrition of rats. Young rats placed on a diet extremely low in fat, and thereby lacking certain essential fatty acids, soon manifest several characteristic abnormalities, namely severe dandruff, scaliness of the feet and tail, retardation of growth, purpura, hematuria and premature death. Administration of very small quantities of the essential unsaturated fatty acids or their esters results in the rapid disappearance of all signs of the fat deficiency disease. No such beneficial effects follow the ingestion of saturated fatty acids even in large amounts. Similar observations were reported by Hansen and Wiese. When very young dogs were given a diet low in fat (0.13 per cent) which furnished only 1 per cent of the total calories but which otherwise normally included all the necessary vitamins it was noted after about three months that the skin of the animals became dry and the hair dry and coarse. This was followed by a definite flaky desquamation with large scales and fine scurfy

specks appearing over the entire body, but most marked on the ventral surface. The skin and hair of the litter mates receiving 28 per cent of their calories as lard in the diet remained clear and soft. Coincidental with the skin changes, marked differences in the degree of unsaturation of the fatty acids of the blood serum were demonstrated. The most marked change in the iodine number of the fatty acids was found in the acetone soluble fraction the average value being 83.9 for the animals receiving practically no fat, in contrast to 118.7 for the dogs getting lard in the diet. These changes in animal skin and hair, due to deficiency of unsaturated fatty acids, have not been demonstrated in human beings (see Squalene, page 54).

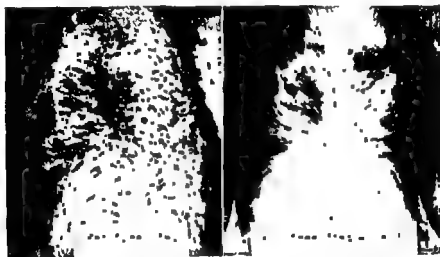


Fig. 69—Influence of dietary fat on the skin of dogs. (Courtesy Urbach. *Skin Diseases, Nutrition and Metabolism*. Grune & Stratton. Original photograph by Drs. A. E. Hansen and H. F. Wiese.)

Proteins are known to have certain effects on the scalp and hair. Amino acid and protein preparations may be conveniently divided into two general classes: (1) mixtures of those amino acids considered essential to human nutrition that are used to combat protein deficiency imposed by severe illness or starvation, (2) individual amino acids that may be used for specific therapeutic purposes.

Preparations in the first class include (a) hydrolysates of protein or sources of protein prepared by various methods of artificial digestion designed to provide adequate amounts of the essential amino acids and (b) mixtures of synthetic amino acids. Preparations in the second class include amino acids such as aminoacetic acid (glycine).

Keratin contains a higher proportion of cystine than is found in most other proteins and thus a higher content of sulfur. Where there is a considerable degree of scaling from the scalp and body (exfoliative dermatitis) it may be necessary to increase the ingestion of sulfur-containing amino acids (cystine, methionine). Specific implications with reference to hair growth and protein nutrition in man are not possible as yet although Hegstad reported an increased need in chickens

for certain amino acids (glycine, arginine) when rapid feathering was a breed feature Urbach also described a characteristic dermatosis which makes its appearance in young rats kept on a diet in which the source of protein consists essentially of unheated and uncoagulated commercial dried egg white. The initial symptoms consist of a dermatitis which is at first confined to the groin, genitalia, neck, and the area around the mouth. As Gyorgy pointed out, this is almost completely analogous to intertrigo in infants, which is commonly one of the first manifestations of seborrheic dermatitis. After this initial phase, brown, adherent scales of varying size and thickness make their appearance, being generally confined to the back. Gyorgy stresses the similarity between these lesions and the "cradle cap" of seborrheic dermatitis in infants (page 238). The pigmentation of the scales is a result of their high fat (cholesterol) content. In later stages, there is a generalized erythematous, scaly, greasy, pruritic dermatitis which resembles the exfoliative seborrheic dermatitis seen in adults or its counterpart in infants erythroderma desquamativum (Leiner's disease). The dermatitis involving the eyelids of the animals creates a picture commonly known as "spectacled eyes". The epidermis of the rat is shed in thin scales, large and small, which, because of their rapid and continuous production, become devoid of fat and therefore colorless. Generalized alopecia often occurs. In black and piebald rats the coat shows signs of depigmentation, the hair turning brownish or even gray. Microscopic examination reveals extensive hyperkeratosis, some parakeratosis, acanthosis, and edema. There is an excessive amount of sudanophilic fat in the hyperkeratotic lamella, according to Sullivan and Nicholls. Administration of biotin results in restoration of the integrity of the skin as judged by gross and microscopic inspection. Recent studies, as yet unconfirmed, indicate that ingestion of large quantities of gelatin may also influence hair fragility and growth.

Vitamin deficiencies may affect the scalp. The term vitamin A has been applied to several substances and mixtures of these substances which produce a specific demonstrable physiologic effect. There are at least five substances which produce this characteristic response in the animal body. These are vitamin A itself, and its precursors, alpha, beta, gamma, carotene and cryptoxanthin. The precursors of vitamin A are produced in plants, and in most animals ingestion of these substances results in the formation of varying amounts (depending on the species of animal and the precursor fed) of vitamin A.

Changes in the hair and nails may be observed in cases of severe avitaminosis A. According to Pillat, the changes in the hair may be listed in the following order depending on the severity of the case: first, the hair loses its sheen and luster and shows signs of dryness; then, if the avitaminosis becomes more pronounced the hair begins to fall out or to blanch. These changes are apparently caused by atrophy of some hair bulbs and by cystic degeneration of others.

The principal microscopic changes in the skin and scalp due to vitamin A deficiency consist of superficial hyperkeratosis of the epidermis extending into the mouths of the pilosebaceous follicles. Subsequently this follicular hyperkeratosis becomes so pronounced that the orifices may become partly distended by a bulbous mass of keratin although the hair continues to grow and the sebaceous glands may

remain normal. If the keratinization progresses further, the follicular orifices become widely distended and the growth of hair and the outflow of sebum are definitely impeded. Eventually this process leads to the formation of a distinct plug consisting of a stratified mass of keratin, the hair follicles then become completely disorganized and the attached sebaceous glands atrophy. Steffens and associates succeeded in producing experimentally the microscopic cutaneous lesions of vitamin A deficiency in a healthy human subject, and Moulton, Sullivan and Evans, and others have induced keratotic plugs in the hair follicles of rats. Moulton points out, however, that cessation of hair growth and atrophy of individual sebaceous glands are caused, primarily, by the mechanical obstruction presented by the keratotic plugs that fill the hair follicles.



Fig 70—Hyperkeratosis of a hair follicle in vitamin A deficiency (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph by Dr. Chester N. Frazier.)

On the basis of the clinical and histopathologic similarity between these dermatologic lesions and other dermatoses characterized by follicular hyperkeratosis, it has been claimed that keratosis pilaris, lichen spinulosus, and ichthyosis follicularis are merely descriptive terms for the same cutaneous manifestations of avitaminosis A. Moreover, there is some evidence that other hyperkeratotic, parakeratotic, and dyskeratotic conditions, such as keratosis follicularis (Darier's disease), pityriasis rubra pilaris, ichthyosis folliculitis keloidalis, callosities, keratoderma of the palms



Fig 71 —Photomicrographs of sections of skin from rats maintained from birth on a diet deficient in vitamin A. Note the progressive keratinization and distention of the follicular orifice. A Rat killed at age of 23 days. B Rat killed at age of 55 days. C Rat killed at age of 85 days. (Courtesy Urbach. *Skin Diseases Nutrition and Metabolism*. Grune & Stratton. Original photograph by Dr. F. H. Moulton.)

and soles, keratosis blennorrhagica, xeroderma pigmentosum, leukoplakia and kraurosis vulvae may be caused, at least in part, by a dysfunction in vitamin A absorption, transportation, and utilization.

Hypervitaminosis A may also affect the scalp. Early signs of excessive vitamin A intake include sparse, coarse hair, loss of the eyebrows, and a scaly, rough skin. Sulzberger and Lazar reported a case of hypervitaminosis A in an adult. In addition to the other changes considered typical of this syndrome, their patient manifested coarse, brittle, and dry hairs in the scalp, axillary and pubic areas. The hairs were easily removed from their attachments and those comprising the eyelashes and eyebrows were almost entirely gone. One of the most striking features of their case



Fig. 77 — Avitaminosis A with keratosis pilaris type of lesions affecting neck and occipital region of scalp. (Courtesy Dr. F. Reiss.)

was the undeniable resemblance between the skin changes presented by this hypervitaminotic state and those characteristic of hypovitaminosis A. A partial explanation of this paradox was advanced by these observers in their comments concerning the relationship between the thyroid gland and vitamin A. Most persons with hypothyroidism also have low vitamin A levels and high carotenoids, and it is presumed that thyroid activity is needed for the conversion of carotene to vitamin A. Sulzberger and Lazar may be justified in their postulation of high lipid levels and hypothyroid symptoms. Of interest also is their postulation that thyrotropic hormone may be counteracted in some manner by vitamin A.

Vitamin B deficiencies may show mild, associated scalp changes. Ariboflavinosis is often associated with sebaceous dysfunction and the oil glands react with increased oiliness and scaling of the sites involved in the seborrheic state (nasolabial folds, scalp margins, ears, sternum). Later on, filiform excrescences and sebaceous plugs may be observed at the same sites. The effects of pantothenic acid and para aminobenzoic acid on the hair are discussed under the heading of Pigmentation (page 76), and the changes reported following use of vitamin B₁₂ are mentioned under the heading of Seborrheic Dermatitis (page 242).

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CHAPTER II

NORMAL SCALP, HAIR PREPARATIONS, DERMATITIS

1. CARE OF THE NORMAL SCALP

Care of the normal scalp and its hair is the day to day attention which we give our scalp and hair to maintain its health and to increase its beauty. This definition would therefore not only include the usual brushing, combing, and cleaning, but also the cutting and waving, the application of lotions, fixatives, and straighteners which have been developed to satisfy the hair fashions of the day.

So many questions have been raised about the values of the many things done to the scalp and hair, that it seems worth while to recapitulate under a single chapter heading the sum of our knowledge about these subjects even though much of it might appear elementary to the trained dermatologist. Frequently the truths which seem most evident to the physician are those which he fails to call to the attention of his patients, erroneously believing that everyone must know as much as he about such everyday subjects. Nothing could be further from fact. A mass of old wives' tales about the care of the hair is our natural heritage and only the replacement of these fairy tales with the discoveries of modern scientific experimentation will serve to free us from prying on such fantasies.

BRUSHING

The implement chosen to brush the hair is just as important as the manner in which the brushing is to be performed. The one characteristic which these brushes should have without fail is the ability to undergo frequent cleaning without injury. The bristles should be well spaced to allow washing to free accumulations of dirt *at their base and should be set in frames able to withstand soap and water.* Lacquered or polished wood is not satisfactory because repeated immersion causes the varnish to disappear, leaving the porous grain to absorb foreign matter. Metal frames are preferable though unattractive but best of all are the ones more recently manufactured of pastel colored plastics which seem capable of taking all kinds of physical punishment. Brushes should be washed in soapsuds and water regularly whether they show signs of soil or not. Natural bristles, when immersed too frequently may become soft and too yielding to have any value as a medium for brushing the hair. Nylon bristles are more resistant. They may be wiped dry immediately after washing, while natural bristles must be allowed to dry in the sun or near, but not too near, artificial heat. It is unnecessary to use boiling water for antiseptics of one's own brush when the scalp is a normal healthy one and the brush

has been used by one person only. When frequenting a beauty shop, however, one should insist upon the use of a sterilized brush which is brought into the booth wrapped in a sterile container. Since one prevalent theory is based on the premise that dandruff may be transmitted by a microorganism, insistence upon sterile implements is merely a normal precaution and reputable shops have long made this a practice.

Brushing should be performed regularly night and morning not because a hundred brush strokes each time were thought by our grandmothers to evoke luxuriant heads of hair, but because it aids combing by unknotting tousled hair in a gentler fashion than the comb, and because it temporarily affects the circulation of the scalp. The hair should be divided into strands and brushing should begin near the bottom of each strand, which is held firmly between the thumb and forefinger. When a two or three inch sector at the end of the strand seems to have been freed from all snarls, the hair should be grasped several inches higher and brushed down as before. This process should continue until finally the brush is allowed to begin at the scalp and sweep down the entire strand of hair to its end without encountering any tangles. Careful treatment of this nature excludes the breaking and tearing out of individual hairs by too hasty and too violent strains on their tensile strength. Such procedures are, of course, for long hair. Short hair may be brushed directly from the scalp. The therapeutic value of brushing is the exercise which it gives the scalp by pulling at it, no matter how gently. As with massage, this action may affect the peripheral circulation of the scalp by producing temporary vasodilatation. Recent observations purport to show that exercise which involves bending or keeping the head in a position lower than that of the rest of the body is just as effective in producing capillary vasodilatation as brushing was thought to be. For this reason lying for periods of time with the feet well above the level of the head causes a temporary redistribution of the blood supply both to the scalp and to the face.

Although brushing of the normal scalp is of some value, it must be modified in diseased states involving the hair. Where the hair is of decreased viability and its attachment is less than normal, frequent and vigorous brushing may increase hair fall and result in further damage to the malfunctioning hair. Under such conditions gentle massage of the scalp and the discontinuance of daily, vigorous brushing are advised.

COMBING

Combs are employed not only for unsnarling tangled hair but also for the essential parting of the hair and its arrangement. No matter of what material the comb is made—whether ivory, shell, metal or plastic—the teeth should be evenly spaced and separated sufficiently to make thorough cleaning between the teeth not too arduous a task. It is important to see that the teeth have rounded, blunt ends to avoid accidental scratching of the scalp since we now know that many diseases of the scalp would not have been transmitted without an initial traumatic excoriation or abrasion which simplified the subsequent processes of irritation, sensitization and infection.

Since combs are used even more frequently than brushes, both men and women carrying them about in pockets and pocketbooks, it is most important that their cleanliness be maintained and that no borrowing whatsoever be tolerated. As with brushes, soap and water are adequate detergents. A small nailbrush may be used to clean between the teeth to be certain that all grease, dandruff scales, and city dust and grime have been removed. Sterilization may be assured by using weak solutions of alcohol or ammonia.

CUTTING

The only reason for cutting the hair is to add to one's comfort and to keep up one's appearance. The old fashioned notion that frequent cutting of the hair and even shaving the scalp would augment its thickness has long since been completely discarded. The source of this fallacy must have been the gardener's successes at cutting back rosebushes and other shrubs to make them grow more successfully. The hair of the head which the naked eye discerns is fully developed, so no shaving or cutting can possibly affect the rate of its growth or influence its thickness. Only elements which reach the hair while it is still in its follicular sheath can affect its health. Both Bulliard and Trotter went through painstaking research to prove the scientific fact with which they were both perfectly well acquainted: no part of the hair shaft above the surface of the scalp is capable of independent growth. All growth develops within the follicular sheath. Perhaps the misconception arose because the rate of growth is more easily observed after cutting the hair short, but Strangeways after careful timing and measurement, also concluded that the ultimate length and texture of hair remains unaltered by simple cutting.

SINGEING

When the ends of otherwise healthy hairs split, there is no cause for concern. The multiple concentric layer structure of each hair with its cortex, medulla and horny outer sheath may separate at the end each set of cells forming its own termination divided from the others. If this is objectionable it is only necessary to cut the hair shaft above the section which has already shown signs of separating. There is no scientific value whatsoever in singeing the hair with a wax taper. It is true that the flame eliminates the split end but it also chars the hair shaft and tends to dry it higher up. Microscopic examination reveals the charring of the inner layers of the hair far above the point where the cuticle has been singed.

The unscientific notion that singeing is preferable to cutting was originally based on the erroneous belief that the entire hair is constantly nourished by a life giving fluid which flows through a hollow canal in the hair shaft. These pseudo scientists thought that cutting would open an end of the canal and through this orifice the nourishing fluid would be lost with resultant death of hair. Singeing they believed would weld the end into a closed terminal point which would act as a stopper to the vital nutritive elixir. Since as stated above the hair shaft with which we are dealing is to all intents and purposes a matured appendage beyond the scalp surface and not an animated one this whole theory is nothing but primitive folklore.

SHAMPOOING AND WASHING

Shampooing was originally intended for the purpose of cleansing the hair and scalp, and this should still be its primary objective. However, when one reads the advertisements for a constantly growing list of new shampoos and ones 'brought up to date,' we read not of their detergent and grease dissolving value, but of their ability to leave the hair with a lustrous sheen. In other words, instead of just cleansing the hair shaft of all foreign matter, the advertisements brag that the shampoo adds to the glory of lady's tresses. Actually, the new synthetic detergents now being used in shampoos are such efficient cleansing agents that they actually remove the natural oils in the hair too completely. To prevent this over-cleansing, various oils, lanolin, and egg yolk are added to diminish the detergency of the shampoo. The proper treatment is to cleanse the hair and scalp first and then, after the shampoo is completed, to add to the hair an appropriate oil to put the hair back into condition with regard to manageability and luster.

There are always questions about the advisability of frequency in washing the hair, particularly hair which is characterized as 'dry.' As a general rule, once every five days is ample when the hair is not excessively oily, although the more often it is washed, the better it is for the hair and the scalp. Some believe that once every ten days is sufficient for the greasy head, and once every two weeks for the dry. If dried properly, and subsequently treated according to the type of scalp, it is believed that the hair may be washed with impunity as many times as one has the energy to do so. Proper drying does not mean rubbing the wet hair briskly with a towel. Experimentation has shown that the tensile strength of hair when wet is considerably lessened, so rather than risk breaking the hair, it should be exposed to heat, preferably that of the sun although a warm current of air propelled by the fan of a hand dryer is also adequate. The inquisitional dryers which completely cover the head must be carefully regulated since overheating will injure the hair by excessive drying, rendering it brittle, with the danger of damage when exposed to brush and comb.

The ideal shampoo should cleanse the scalp without either irritating the epidermis or causing excessive reduction of its natural oil. A shampoo with too alkaline a reaction or one with too great detergency will dry the hair and make it brittle, instead of leaving it soft and lustrous. Since shampoos are designed specifically to remove all foreign matter, they must not themselves produce, either alone or in combination with water, any insoluble precipitate remaining on the hair shaft. And lastly, for purely psychologic reasons when combined with water they should evoke an instantaneous creamy lather. When developing a shampoo formula it is important to remember that the fundamental objective is a substance which cleanses the hair and the scalp and that no one should expect a product which will effectively ameliorate a diseased condition. All shampoos except those characterized as 'dry' depend upon the addition of water. Hard water should be avoided for shampoos or, if no soft water is available it should be treated until it becomes soft. So called 'hard water' is water with a sufficiently high mineral salt content to interfere seriously with the lathering of soap. If the salts present are merely the carbonates and bicarbonates of calcium boiling will precipitate them, but if chlorides

and sulfates of magnesium and calcium are components, boiling is no help and the water will remain "permanently hard." When this type water is employed, it precipitates the soap because of the chemical interaction of the calcium and magnesium with the fatty acids forming insoluble soaps. They leave deposits on the hair shaft, thereby dulling the hair, and the scalp becomes irritated by the action of these precipitates.

When soft water is totally unavailable, the permanent type of hard water may be softened at home by distillation. A Permutit system for home use has also been made available, but the simplest expedient is to add a 1 per cent solution of Calgon, a sodium hexametaphosphate. To test whether the water supply is soft or hard one need only to shake up the same amount of soap solution in two test tubes, one filled with the questionable water and the other with distilled, and compare the subsequent lathering. For the water to be sufficiently soft to use in shampooing, the lather should persist for a minimum of two minutes after agitation.

The best sources of soft water are rain, which is usually pure but may become contaminated passing through smog or being collected in unclean receptacles, and distilled water, which is the condensation of steam arising from boiling water. Water from subsurface springs which becomes purified as it wells through the soil, which acts as a filter, and water from artesian wells may have varying degrees of hardness, and whereas it is pure treatment may be required to soften it.

2. HAIR PREPARATIONS

The number of preparations designed for use on the hair and scalp is so great that it would be necessary to devote an entire volume to complete coverage of the subject. Accordingly, I have attempted to confine this chapter to a discussion of those preparations both in widest usage and of greatest importance to the physician. Typical examples and formulas of the various types of hair preparations are listed in the formulary contained in the Appendix of this book. The reader interested in the minutiae of the chemical, pharmacologic, and cosmetic formulation of the various products is referred to the excellent texts of de Navarre, Harry, Thomsen, Poucher, Winter, Goodman and others.

SHAMPOOS

Three types of shampoos have been developed, all obtainable in liquid or solid forms. These are the soap shampoos, the dry shampoos and the soapless shampoos.

Soap Shampoos

A shampoo in powdered form is composed of varying components such as foaming agents, saponin, together with such alkaline materials as sodium carbonate, borax, sodium sesquicarbonate and trisodium phosphate. These compounds facilitate saponification of the sebaceous secretions and act as water softeners at the same time. The product may be scented with various oils such as lavender and rosemary, or be perfumed by eau de Cologne and the artificial musk, xylol. Sometimes henna powder up to 5 per cent of the total bulk is a component added to highlight dark hair, and the dried flowers of camomile are supposed to give comfort to blondes, but this is highly problematic.

Soap shampoos in liquid form are far more popular because they are simpler to handle. These liquid shampoos are prepared in two ways. Either suitable oils are saponified with an alkali and then diluted with distilled water, or a quantity of soft soap is dissolved in water or in a mixture of water and alcohol. One of the more popular is coconut oil, because its soap lathers luxuriously and forms a clear solution when diluted with water. Liquid soaps made of coconut oil are quite drying and irritate some scalps. Olive oil soaps are preferable from a dermatologic point of view, but used alone they do not lather very well. Soaps made of a combination of the two oils seem to be a particularly happy solution. Other natural oils which may be successfully employed are almond, arachis, castor, cottonseed, linseed, palm kernel and palm nut, peach kernel, and soy bean oils. The alkaline content is usually provided by potassium, sodium, or ammonium since these form the only salts soluble in water.

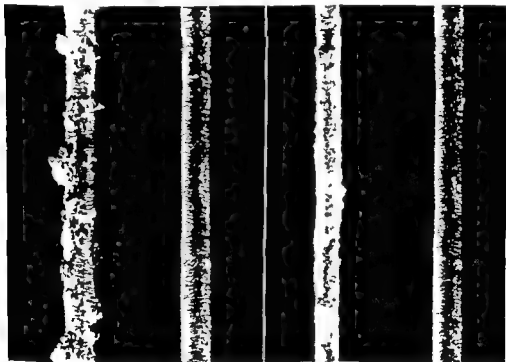


Fig 73

Fig 74

Fig 73—Left Hair coated with dirt and grease. Right Same hair following shampoo (Courtesy Mr R. Reed and the Toni Co.)

Fig 74—Left Hair cleansed by means of bar soap. Right Same hair following liquid shampoo (Courtesy Mr R. Reed and the Toni Co.)

Dry Shampoos

Dry shampoos are so called not because they are not liquid, but because no water is present in their composition. They are actually dry cleaners for the hair and usually contain carbon tetrachloride or alcohol both of which dissolve fats.

Because the hair dries rapidly as the solvent evaporates after the grime has been removed with a towel, such shampoos are particularly suitable for invalids. Repeated usage, however, might prove harmful since industrial methylated spirit or isopropyl alcohol may be a scalp irritant on repeated use. The fumes are also quite toxic on inhalation. Skilled operators are required to handle the liquids, which are excellent solvents, but they should be recommended for infrequent use only in special instances.

Egg Yolk Shampoos

One of the very few folklore formulas which has proved itself effective when checked by modern science is the egg yolk shampoo of our grandmothers. Egg yolk contains cholesterol, a substance thought to be of value in maintaining the quality of hair. The egg yolk has another advantage—being an excellent emulsifying agent, it serves to emulsify the oils on the hair with water thereby permitting them to be readily removed by rinsing.

Soapless Shampoos

In comparatively recent years new types of detergents have been developed which are used in shampoos. These substances may be divided into two broad classes—the salts of sulfonated fats or oils, such as the ammonium salts of sulfonated castor oil, and the salts of sulfonated fatty alcohols such as the sodium salt of sulfonated lauryl alcohol. The latter types are newer and have some advantages over the sulfonated fats. Both types have a distinct advantage in that their calcium and magnesium salts are soluble and therefore may be used with hard water. The fatty alcohol type may be used in a neutral or even acid solution. The main disadvantage of shampoos made of such substances is that they are so efficient as detergents that they actually clean the hair too well, removing almost the last trace of oil. This leaves the hair dry, brittle, and harsh. To obviate this occurrence, various substances such as egg yolk and oils of various types are added to the shampoos, their main function being to decrease the detergency in order to leave in the hair a residual amount of the natural oils.

The commercial category of 'sulfated fatty alcohols' or sodium alkyl sulfates is a misnomer. They are not true sulfonic derivatives of the fatty alcohols but rather sulfuric acid esters as evidenced by the formula RCH_2O-SO_3Na .

Sodium lauryl sulfate is being marketed at half strength to add to its bulk. The customer actually needs only one fifth of an ounce for a shampoo but would be discouraged to see his money being spent for so small an amount of powder. In order to satisfy the unschooled patron, the bulk is increased to half an ounce per shampoo by adding some harmless ingredient as a diluent. Just as the customer requires a minimum bulk for his money so does he require good lathering for a shampoo. Psychologically the consumer has been convinced by years of advertising that it is the lather which does the cleaning—which is relatively true about soap but totally unrelated to the detergent properties of soapless shampoos. Since sodium lauryl sulfate is less than 1 per cent soluble in cold water it is really not satisfactory for a liquid soapless shampoo. The problem has been solved by utilizing the viscous yellowish brown triethanolamine salt of lauryl sulfate in a concentrated aqueous solution with water and perfume.

In addition to the above, Sapaminc, an acid soap, has been developed from the higher fatty acid in conjunction with ethylene diamine, i.e., diethylamine-ethyl-oleylamide $[\text{CH}_2(\text{CH}_2)_7, \text{CH}=\text{CH}-(\text{CH}_2)_7-\text{CO}-\text{NH}-\text{CH}_2-\text{CH}_2, \text{N}(\text{C}_2\text{H}_5)_2]$, a less expensive product with effective emulsifying properties.

The sulfonated oils are a group of oils which have been treated with sulfuric acid or other sulfonating agents and then neutralized. Sulfonated castor and olive oils are usually included and prove to be good wetting agents, detergents, and emulsifiers. The mixture of these sulfonated oils is preferable to either one used alone since castor oil is too sticky and the sulfonated olive oil is too thin. One of the main objections to the sulfonated oils in contradistinction to the sulfonated alcohols is that they produce practically no lather. These preparations are often referred to as soap substitutes because they are well tolerated by the sensitive skins and scalps of individuals who are unable to use ordinary soaps.

In looking for chemical products which would not have these faults, the condensation products of high molecular albumins combined with high fatty acids proved to have remarkable detergent and foaming characteristics. There is one soapless product which reacts slightly acid, $\text{pH} = 6.0$. It is not too exaggerated a degreasing agent, leaving the hair in a soft, silky condition. Another, having a pH of 9 plus, may be used with liquid soaps for shampoos to increase the resistance of the latter to lime soap precipitation.

Another method of avoiding precipitation of calcium soaps in hard water by ordinary soap, is to add a solution of sodium hexametaphosphate which forms a complex with calcium, thereby effectively removing the calcium. Because of these chemical actions, sodium hexametaphosphate will soften water without any precipitation whatsoever and will dissolve any insoluble calcium or magnesium soaps which might have formed as a temporary scum.

Shampoo Rinses

Soft water is an ideal rinse. Many women insist upon vinegar and lemon, believing that they soften the hair. They are both helpful but for an entirely different reason: their inherent acidity counteracts the alkalinity of the shampoo and helps the scalp regain its normal acidity. As a hair rinse, sodium hexametaphosphate is of value as it dissolves any precipitated calcium or magnesium left in the hair shaft by soap shampoos. As a water softener, this product should be used in quantities of 2.5 ounces per degree of hardness for 100 gallons. Naturally, the soapless shampoos require no special rinsing at all. An added undercurrent inviting use of these rinses has been the manufacturers' sound idea of combining them with herbs which brighten the hair, such as camomile for fair hair or an infusion of henna leaves to give reddish highlights to brown hair. The latter only dyes the keratin.

shampoo
of tarta

HAIR LOTIONS

So many erroneous and fantastic claims have been made by manufacturers of hair lotions that there is a tendency among intelligent persons to deride their use under any circumstances. Actually the dermatologist will admit that there should be

a market for a good antiseptic conditioning and stimulating lotion. The method of application involving friction is probably the basis for many of the good results claimed by the advertisers of most preparations. The extra encouragement given by massage to the blood supply of the hair follicles may temporarily increase their nutrition and thus temporarily benefit the hair papilla, but the entire premise remains in the realm of pure theory. It is perfectly possible that such massage for two minutes a day night and morning will do almost as much good without a lotion as with one.

Antidandruff lotions are merely antiseptic lotions preferably mild and non irritating which keep the hair and scalp as bacteria free as possible. The lotion must be used continuously since the treatment is in all cases a temporary one and requires constant reapplication. The bases must differ of course, depending on whether the lotion is to be used on a scalp which may be characterized as "dry," or on one known as "greasy." The latter requires an alcoholic base, the former an oily one. The notion that if enough oil is present in a spiritous lotion it will prevent undue drying of the hair is completely incorrect. It is the total absence of water in these dilutions which exerts an exaggerated dehydrating effect on the scalp. Therefore to prevent drying water should always be present in such lotions as well as enough oil to lubricate the keratinous layer whose natural oils may have been removed by the application of aqueous alcohol. Because fats are not readily soluble in lotions containing water it may be more effective to formulate an antiseptic emulsion having a small amount of oil and a neutral or slightly acid base.

The types of lotions listed below are all antiseptic reconditioners but none of them is capable of growing hair for the simple reason that there is as yet no known preparation which will effect such a miracle. When applied with enthusiastic friction they help to free the hair mechanically from dandruff and to render the scalp temporarily semisterile making it less liable to bacterial infection. Persons with oily scalps may use them frequently but those with dry scalps should be careful because most lotions may be too drying for normal hair. They may be used from time to time for their stimulating effect provided they are alternated with applications of brilliantine or animal or vegetable oil (see Chapter X, pages 552-554).

Hair lotions usually contain an antiseptic and a mild rubefacient. There are many drugs employed in the care of the scalp the chief of which are sulfur, salicylic acid, quinine, formalin, resorcinol, resorcinol monoacetate, oxyquinoline, oil of cade, betanaphthol, capsicum, pilocarpine, cantharides, isopropyl alcohol and cholesterol.

Sulfur is a frequent ingredient of many hair preparations. On the scalp it is employed primarily because of its antiseptic properties.

Salicylic acid is also a favorite remedy often incorporated in various scalp preparations. It is mildly antiseptic and antipruritic in effect. When a keratoplastic effect is desired the concentration should never exceed 6 per cent inasmuch as the drug has a keratolytic action in higher concentrations.

Quinine is effective as a bacteriostatic agent and mild rubefacient so that it keeps the hair in a relatively sterile condition. It should be used with caution inasmuch as many persons are allergic to this drug despite this fact it is employed in many types of scalp wash.

Formalin is a 37 to 41 per cent solution of the powerful germicide and irritant, formaldehyde. A 0.5 per cent aqueous solution is sufficiently strong to eliminate most microorganisms from the skin and scalp.

Resorcinol, which is m-dihydroxybenzene, is less toxic and irritating than phenol (which should not be applied frequently to the scalp) and more effective as an antiseptic. It also has astringent and antipruritic properties. It can never be used on hair which has been shampooed with soap or alkali because it acts chemically, producing a discoloration of blonde hair. It also turns gray hair a greenish yellow shade. Resorcinol monoacetate, a reddish yellow viscous liquid, soluble 10 to 30 per cent in acetone combined with 11 to 10 per cent of a solution of one part monoacetate in two parts acetone, combines with formic acid and water to make another antiseptic hair tonic. It does not discolor hair and is less irritating to the scalp than resorcin. Oxyquinoline is used also for its bactericidal action. Various fungicides are being incorporated in some of the newer formulae.

Juniper tar oil, known as oil of cade, is not so successful with the public because of its muddy color and unpleasant odor. One half to two per cent may be used in an oil or ointment base, either as a simple oily mixture or as an oil in water emulsion for scaly, psoriatic scalps. Beta hydroxynaphthalene is a powerful germicide much less toxic than phenol. It is difficult to formulate because it is only slightly soluble in water though improved through the addition of boric acid. This reducing agent must be used with caution because higher concentrations are keratolytic and prone to irritating action.

Many lotions employ various concentrations of tincture of capsicum while others use 4 per cent of the alkaloid contained in jaborandi leaves known to us as pilocarpine. Another lotion makes use of dried *Cantharis vesicatoria*, containing an irritating crystalline lactone cantharidin, only 0.002 per cent of which is safe to use at one time. Other than mild antiseptic and strong rubefacient properties, these drugs show no physiologic attributes to justify their reputation for the promotion of hair growth.

Isopropyl alcohol or properly denatured ethyl alcohol diluted with water is suitable as the solvent in most hair lotions.

The final concoction has not been made in hair lotions. Harry states that a simple oil and water emulsion containing a proportion of fish oils or unsaturated fatty acids in low proportion or a vitamin concentrate preferably vitamin A might prove efficacious, and that the result of "biodynes in a suitable media of physiologic pH" would bear investigating. A recent preparation (formula A 10, Richard Hudnut) incorporating fatty acids, vitamin A, and several other ingredients is a step in this direction.

SETTING LOTIONS AND LACQUERS

There is a variety of hair lotions and lacquers whose sole purpose is to help fix water waves and finger waves and provide them with an artificial finish which is meant to add formal beauty to the hair. Although the appearance is smooth and neat, it is not perhaps so attractive as a more natural softness. These solutions may contain alkalis and borax or gum substances in alcohol and water. In order

to compete with other beauty products, they are tinted and scented. The alkalis are employed because they soften the keratin of the hair shaft, making it easier to set with combs or fingers. Gum tragacanth, gum karaya, quince seeds, and pectin are all used. The pectin is a complex carbohydrate derived from various fruits and vegetables and creates a very light setting lotion on the acid reaction side which is claimed to give the hair a more lustrous wave. A new product used as a setting aid is sodium alginate.

The difference between a setting lotion and a lacquer is in the thickness of its solution. Lacquers are light enough to be sprayed on the hair for an immediate effect. Usually they are alcoholic solutions of resins. Not all mucilages may be employed in an atomizer since, when they are sufficiently dilute to be sprayed, they fail to assist the waving. Whatever the type, these products are rapid drying and successful in keeping the hair well groomed although at times exaggeratedly so. The new water soluble resins and hexitol derivatives are also on the market. They have improved flexibility and so adapt themselves readily to the most complicated hair designs.

WAVE LOTIONS

Artificial waving which cannot be classified as permanent has been practiced since the earliest recorded times. People have always appreciated the flattering more easily handled frame for the face which waving provides. Wetting the hair and wrapping it around metal fabric or leather curlers has long been a woman's method of improving the appearance of her hair. Later the water wave, held in place by combs, or the finger wave held in place by pins replaced curlers. Firm textured and not too oily hair of sufficient thickness takes such a wave set successfully sometimes retaining the wave for days. If the hair is naturally perfectly straight and somewhat greasy, it may lose the wave in a few hours if the atmosphere becomes overdamp or if in summer there is excessive scalp perspiration.

Following the water wave hairdressers developed waving by means of heat and curling tongs. This was possible because normal hair at regular temperature and humidity contains enough water to be used for setting purposes; the keratin requiring the water to start the action which realigns the fiber molecules. Leftwich as early as 1901 tested the effect of curling tongs. After one curling a 7 ounce hair broke at 6 to 6½ ounces. The tensile strength of several other hairs curled daily for a week remained unchanged. Actually hairs which had been wet and stretched tightly around curlers by hand proved to be more fragile. The study was not sufficiently scientific to lead to any conclusions because Leftwich failed to record the degree of heat employed. Since then it has been shown that a safe temperature for curling tongs should approximate 130° C.

The mechanics of permanent waving must deal first of all with strongly alkaline lotions which have been developed to accelerate the heat waving process. These include morpholine triethanolamine monoethanolamine ammonium hydroxide borax sodium and potassium carbonates and bicarbonates.

A satisfactory formula contains ammonium hydroxide combined with borax and sodium or potassium carbonate. This combination gives a successful wave

after only ten minutes' steaming time. Salts such as borax or the alkali carbonates seem to produce a more lasting wave, perhaps due to the fact that they tend to increase the temperature of the hair. Alkaline sulfites seem to possess some specific action on the keratin which accelerates the waving action.

Because of the strong odor of the ammonia in permanent wave solutions, triethanolamine, a viscous, hygroscopic liquid, is often used. It produces a fairly successful wave. Monoethanolamine is more alkaline and even stronger than ammonia. It has the advantage of being colorless but the ammoniacal odor is persistent though less so in dilution. Morpholine is another synthetic compound, stronger than triethanolamine, employed for this purpose. Many of the above formulas contain potassium sulfite. Recently this, too, has been replaced by sulfites of the amino compounds, such as triethanolamine sulfite containing about 10 to 11.5 per cent sulfur dioxide, and monoethanolamine sulfite containing about 10 to 18 per cent sulfur dioxide.

After the hair has been thoroughly dampened with one of the permanent waving lotions described above, the hair is bent into the form of a spiral curl, enclosed in a metal cylinder, and heated to the temperature of steam. After this stage is completed, the hair is allowed to cool and then the curls are combed out. The hair is washed to free it from the chemicals deposited by the lotion. While the hair is still damp, the desired wave or curls are set with combs, finger pressure, and multiple hairpins. It is then secured by a net and dried under hot air.

Even though the resultant wave is called "permanent," it is only comparatively so when contrasted with the earlier water waving, finger waving, and curling tong techniques. The pressure of the pillow or a hat may disturb the wave, and unusual atmospheric dampness makes the hair spring back to its spiral curl, giving a fuzzy effect. Setting lotions must be employed several times a week to help reset the wave with combs or finger pressure. Persons who can afford it make weekly visits to the beauty parlor to have the wave reset by skilled operators.

Many questions are asked about the possible difficulties which might be met during the permanent waving process such as whether a permanent wave will fail to take for any one of the following reasons: pregnancy, menstruation, nervous disorders, recent use of an anesthetic, ingestion of drugs, or high blood pressure. The answer, of course, is that none of these states is capable of affecting the condition of the hair during the waving process because the process affects only that section of the hair which appears above the level of the scalp, and we already know that bodily states of health are able to affect only that part of the hair which is still growing within its follicle. The reasons listed above have merely been used to excuse an unskilled operator's failure to try a test curl before waving. The anesthesia and ingestion of drug "excuses" have been exploded by the successful operation of permanent waving units on wheels which are now in use in many hospitals.

There are certain definite disadvantages to the old style, hot permanent waving technique which crop up now and again. A protein reaction due to alkali and heat sometimes darkens or reddens white hair. Very fine hair too often waved tends to have its keratin cells break and never at any time retains the wave as long as coarser hair. Overheating must be assiduously avoided and skilled and experienced operators are the first essential to a successful wave, carelessness may

lead to severe burns of the scalp due to steam generated during the heating cycle. Women to avoid the expense of rewaveing often do not brush or comb their hair for days on end. This lack of normal friction and massage results in the accumulation of scales and dirt on the scalp and cannot help but interfere with the luster and appearance of the hair.

Cold Wave Preparations

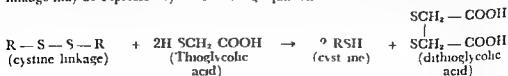
When the so called cold wave process was developed which requires neither electrical heat nor chemical heating pads permanent waving became revolutionized. It made hair waving possible in the home by those who wished to save the money constantly expended in beauty parlors. The cold wave process both in the home and beauty parlor has become the most popular method of waving the hair, as is indicated by the annual sale of millions of units.

The chemical action of the waving lotions at room temperature produces the wave. Fortunately these changes are only temporary, and even after the hair has been exposed to such a treatment for three or four times its cystine content remains unaltered. This is substantially true of its length, tensile strength, elongation and stiffness all of which have been assayed before and after waving by precision measurements.

The chief ingredients used in the popular cold wave preparations are the various alkaline salts of thioglycolic acid. The intensity and speed of its effect are controlled by the concentration of the thioglycolate and the alkalinity (pH). To facilitate application as well as to accelerate the action wetting agents are incorporated and for cosmetic appeal perfumes and clouding agents are added.

Since the cold waving method creates morphologic, physical and chemical changes in the hair it is important to understand what actually takes place when these changes are in process. The fibrous proteins of the stratum corneum which normally are unaffected by the usual chemical actions to which they might be exposed are susceptible to the action of alkalis and to keratolytic agents. Cold wave lotions therefore contain a keratolytic agent with enough alkali, either ammonia or sodium hydroxide and sometimes both to adjust the pH to 9.0-9.5.

The chemical change following the application of such lotions involves the reduction of the cystine to cysteine, rendering it considerably more pliable than normal. The change produced will depend on the strength or concentration of the active ingredients in the solution, the time of exposure, temperature and pH. The alkaline salts of thioglycolic acid dissolve hair or wool actively at pH 11.0. At pH 9.0 to 9.5 the usual range produced in cold wave solutions, keratolysis occurs but at a much slower rate. The reducing action of the thioglycolic acid on the cystine linkage may be expressed by the following equation:



To increase the viscosity of the lotion in order to decrease the amount of fluid reaching the scalp and to assist in the physical application of the solution to the

hair, gums and resins are added. Synthetic gums and resins may become sensitizing agents for the operators as well as for the persons on whom they are used. Esthetic effects are secured by coloring agents and perfumes. The "neutralizers," which not only arrest the action of thioglycolate before any damage is done but oxidize cysteine back to cystine, are weak oxidizing agents employed in a weak acidic medium. Peroxides, perborates, bromates, and at times a solution containing citric acid are employed.

With the widespread and increasing use of new products of this type, it is desirable to inquire into the possibility of any toxicologic or dermatologic effects. In fact, numerous papers have been published reporting, in some instances, alleged manifestations of allergic sensitivity and even of systemic effects.

A large amount of work has been reported on the toxicity of thioglycolates. Unfortunately, in the majority of the reports, specific information as to chemical composition, concentration, alkalinity (pH), dose and duration of application is lacking so that comparisons are difficult. These data are of primary importance in determining the toxic effects of the thioglycolates. For example, the alkalinity plays a major role in so far as irritant effects are concerned. Furthermore, systemic toxic effects from topical application are proportional to the concentration and to the total dose of the thioglycolate solution as well as to the length of time over which it is applied. Draize has shown that a dose which he characterized as "the human dose," but which actually is far above that used on human beings, when applied daily to the shaven skin of rabbits for 90 days proved to be nontoxic. These studies are only of heuristic importance to the toxicologist because in normal practice the lotion used for waving the hair is applied only once every three to six months and not daily. Furthermore, the whole dose does not touch the skin but on the contrary every effort is made to avoid such contact. Consequently, in so far as an analogy with the rabbit may be made the amount of thioglycolate used in hair waving should prove quite innocuous as far as systemic toxicity is concerned. Experimental evidence obtained on individuals in inordinate contact with cold wave lotions has corroborated these conclusions.

Thioglycolates probably owe their hair waving activity to the sulfhydryl group. There is ample evidence that the sulfhydryl group is of great physiologic importance. It may act as a detoxifying agent. The metabolism of certain drugs and toxic substances involves a combination with sulfhydryl groups present in the proteins of the tissues available as glutathione, cystine, methionine, and homocystine. It is not surprising therefore by virtue of their sulfhydryl group content that salts of thioglycolic acid should prove to be of low systemic toxicity. Indeed the toxic actions of heavy metals including arsenic may be due largely to their combination with the sulfhydryl groups of the proteins of the tissues with the formation of mercaptides. In this connection one may point to the success of dimercaprol, the British Anti Lewisite (BAL), a dimercaptopropanol in the treatment of heavy metal poisoning. Conversely the capacity for metal mercaptide formation of sulfhydryl groups when present in excess may result in unfavorable toxic effects by combination with the metal ions of the metalloprotein enzyme systems.

Dermatologic reactions attributed to cold wave lotions were first reported in 1914. Since that time additional reports of both cutaneous and systemic effects of

TABLE I
SUMMARY OF RESULTS FROM PATCH TESTING OF CLINICAL CASES APPEARING IN THE LITERATURE

Author	CLINICAL DATA			PATCH TEST DATA			pH	Results
	Number of Cases	Diagnosis of Case	Number of Controls	Method of Testing and Time of Exposure	Material Used	Concentration Used		
J. P. Howell (24)	2 women (1 cold wave)	Erythematous contact dermatitis	3	Patch tests	Jampoo Frellida lotion Laving compound Macfarlane's Misture	15 2 15 2 15		neg neg neg neg neg
L. W. Cotter (19)	1 hairdresser	anxiety and post cephalic		Patch test	4 thioglycolic acid (pure) on 10% vit. B ₁₂ oil	1 drop of undiluted oils to 100 vol. of water		pos
	1 hairdresser	post cephalic						
	1 hairdresser (1 cold wave)	anxiety and post cephalic		2 Patch tests				pos
	1 housewife (1 cold wave)	anxiety and post cephalic		Patch test		High dilution		pos
	1 housewife (1 cold wave)	anxiety and post cephalic						
C. V. McGill and L. P. Fry (20)	2 women (1 cold wave) (2 cold waves)	dermatitis		Patch test	Parm wave oils	1:100 dil.	.	neg
C. H. Bock (26)	2 hairdressers	eczema	100 F 20 H	Patch test	Thioglycolic acid buffered with pH 0.6	66	9.0	2 derm. controls Both pos. 17 pos. 45 H pos.
H. Bown (23)	2	dermatitis						
L. G. Brown (27) et al.	12 hairdressers	contact dermatitis	109 women (30 negative)	Stretch tests Patch tests (on 28 mil.) Patch tests	Ammonium thioglycolate Commercial cold wave			2 pos. stretch tests 11 pos. patch tests negative "Jampoo" elicited more positive reactions than washing solutions

name	1159) human (blonde brn. native European syndromes of rumble)	Peter test to confirm with scratch tests	Thiolactic acid	up to 11"	to a primary irritant in low concs 20 (was changing to broken skin) no positives
		Pat. 6 test Contact period h to 96 hrs	soln as positive thiolactate (stabilized or unstabilized)	3 30 thiolactic acid content	
		patch test	butfered oil thiolactate (stabilized or unstabilized)	6 60 thiolactic acid content	Under 4 hrs no positives longer exposures occasional positives no positives
		Pat. 6 test	oil type of oil	allied as for 40	no positives
		patch test long contact	oil thiolactate (aged for concn of 12 to 14)		slightly more irritating than fresh solutions
		patch test h to 6 hrs contact	thiolactic acid (aqueous solution)	6 60	positive responses
		Pat. 6 test 4 6 and 9 hrs 24 hrs 48 hrs 72 hrs 96 hrs	soln thiolactate (cream)	7 30	no positives
		14 Patch tests 24 hrs contact	" " " " commercial cold water soln (saturated with chloride)	" " " "	0 4 positive 13 76 positive 10 44 positive 20 31 positive 3 positive (2 for 1 product 1 for another)
		14 Patch tests Subjects 7 10 days after initial tests 24 40 hr exposure			For test of pos itives slightly lower than 14 original series no positives in the absence of a like initial response

TABLE II
SUMMARY OF DERMATOLOGIC REPORTS

Author	Number of Subjects	Method of Testing	Material Used	Concn. Used	pH	Results
H O. Cappel and L. R. Cappel (25)	--- Red-heads blonds and brunettes	Patch test	Ammonium thioglycolate	7.0%	9.6	negative
			Ammonium thioglycolate + dithioglycolic acid	7.0% 0.2%	9.6	negative
			Ammonium thioglycolate + dithioglycolic acid	6.5% 1.5%	9.4	negative
W. O. McNally and R. N. Scull (6)	154 humans (76 skin diseases; 76 normal)	on skin, one year daily for 2 months	Ammonium thioglycolate	Not stated	---	negative
E. K. Hair (13)	13 humans	Denize sleeve modification 1 hr. contact	Thioglycolate com- mercial preparation	0.847 to 0.912M SE	---	3 mild 1 severe
C. Barban (5)	12 humans	Patch test on scarified area	Commercial cold wave lotion Cold wave lotion + fixative fixative	0.2M --- ---	9.3 --- ---	negative
J. O. Graca and F. L. Eurollier (14)	51 humans 20 humans 30 humans	Patch test Patch test Patch test (double dose)	Ammonium thioglycolate Ammonium thioglycolate Ammonium thioglycolate (Commercial preparation)	0.9034 0.93 M 0.93 M	9.2 9.2 9.2	negative negative negative
R. E. Reed, M. DeBaste and F. L. Eurollier (12)	400 employees	Patch test	Thioglycolates	below M	---	1 positive (exposure not stated)
C. P. McCord (16)	1129 humans (blonds, brun- ettes, Negroes; preponderance of females).	Patch test to forearm, some with scratch marks.	Thioglycolic acid	up to 11%	---	Is a primary irritant in low concns. 2.8% (more damaging to broken skin)

waving lotions, as well as the ingredients thereof, have appeared. A summary of these reports, including both clinical and dermatologic studies, is given in Tables I and II.

It is quite evident that there exists a great diversity of opinion concerning the effects of waving lotions and their ingredients. This is due primarily to the use of a variety of materials, comprising in some instances the waving lotions themselves and, in others, the use of isolated ingredients, to the use of various concentrations and pH of the waving materials, to the employment of a variety of methods of testing, to a variation in the selection of test subjects, and to the inadequate testing of subjects who are suspected of showing systemic toxic manifestations.

It is axiomatic that differences in results reported by various observers should stem from some difference in the experimental approach, yet careful analysis of some of the published data fails to disclose adequate reasons for such divergence. For example, Beek, using a 5 per cent solution of thioglycolic acid neutralized with ammonium hydroxide to a pH of 9.0, reported an incidence of skin reaction far in excess of that which we have found in an extensive and closely controlled investigation. Goldman stated that because the actual cold waving process calls for a preliminary shampooing and cleansing of the hair, as well as for shampooing and wave setting subsequent to application of the cold wave lotion, all the materials used during the cold wave process should be included in performing the skin tests. He concluded that "the shampoo was implicated most frequently by the patch tests" in such cases. Indeed, numerous studies show that the solutions of the chemicals used in cold wave processes are of a low order of irritation, and that they infrequently produce cutaneous reactions.

From a study conducted by Cotter on five patients who had in common a history of occupational or cosmetic contact with cold wave lotions, the conclusions were drawn that the cold wave lotions were responsible for the dermatologic and clinical findings, "because it had been possible to demonstrate their lesions in the laboratory." On perusal of the case histories which were published in detail, it becomes evident that despite the common denominator of contact with cold wave lotions, there are many other and compelling reasons why Cotter was not justified in drawing such a definite inference of causal relationship between contact and clinical findings.

A series of studies was performed by the author and his colleagues on normal subjects and on subjects with various skin diseases on persons with no known contact with cold wave solutions and on many in intimate and prolonged contact in order to delineate fully and critically the part played by cold wave lotions on the health of that part of the population most likely to come in contact with these materials. Over 1,200 persons in varying degree of contact with such preparations were used as subjects. Cutaneous reaction was studied with reference to irritancy and sensitization. Primary irritancy of ammonium thioglycolate was investigated and shown to be of a low order even when the concentration was triple that of the solution in common use.

Paired patch tests using the solutions employed in the cold wave process were performed on 286 subjects with various skin diseases. These tests showed that the

cutaneous reactions from contact with or exposure to these materials were of a minimal incidence and sensitizing potential

Patch tests and physical examinations were performed on 931 individuals, many of whom were factory employees engaged in the manufacture of and in constant or frequent contact with cold wave preparations. Not one of these individuals gave evidence of any skin condition or of systemic disease in any way referable to intimate contact with the lotions. A preliminary study of employment statistics had shown this group to be a representative cross section of the population and not a selected immune or hardened group.

Examinations made of the scalps of 174 women undergoing actual waves with commercial waving materials showed no adverse effects. Each of these women previously had had from one to twenty cold waves. Patch tests disclosed a weakly positive reaction in only one individual. It is of interest that this woman was then undergoing her fourth cold permanent wave. No untoward effects resulted from either the last application or from her three preceding cold waves. The data obtained from this study emphasizes anew the precept that skin reactivity to patch testing should be evaluated only in conjunction with actual usage experience. In actual use the composition of the chemicals may be modified by such factors as volatility, stability, and by contact with air, skin, and other materials, thus altering their reactivity on the skin of the subject.

Thorough physical and laboratory examinations of 226 individuals exposed in varying degrees to cold wave lotions failed to disclose evidence of any acute or chronic condition which might indicate the presence of disease of the liver, the kidneys, or the hematopoietic system. In view of the report of hepatotoxicity due to contact with cold wave lotions, special attention was given to tests of liver function. The icteric index was within the normal limits in all cases. Prothrombin time was slightly elevated in ten subjects. Nine of these permitted further studies which failed to elicit any signs of liver disease. These studies included the cephalin cholesterol flocculation, vitamin K response, and bromsulfalein retention tests, and urinary urobilinogen and coproporphyrin excretion studies.

The results of the cephalin cholesterol flocculation tests, which were obtained on the entire group of 226 subjects, were scrutinized critically because the diagnosis of alleged damage to the liver from cold wave lotions in Cotter's report had been based primarily on this procedure. All individuals with borderline positive reactions were subjected to careful physical examination and further laboratory procedures. One subject only had both a 2 plus cephalin cholesterol flocculation and a slight retention of bromsulfalein (12.5 per cent), but she was symptomatically negative and showed no abnormalities on physical examination. With this one exception, the results of these studies were within the normal range and could not be construed as indicative of liver damage. Physical examinations were performed on all available subjects showing any deviation whatever from the normal on any of the laboratory tests. No significant abnormalities were found. This is not surprising, however, in a group of individuals who worked daily and who generally speaking were in good health. In view of these results, systemic toxic manifestations following contact with cold wave lotions should be carefully verified before

being accepted as indicating a causal relationship. Dermatitis, temporary alopecia, and changes in the physical characteristics of the hair have been reported although the incidence of these reactions appears to be of a low order.

HAIR CREAMS AND FIXATIVES

When the hair becomes dull and brittle due to the use of harsh soap, frequent bleaching and dyeing, or overexposure to sunshine, it is customary to relubricate the hair shaft with brilliantines and emulsified hair creams. These brilliantines and emulsified hair creams, as well as mucilages made from various gums, are used for fixing the hair in the position in which it has been dressed.

Brilliantines usually are made of liquid paraffin (specific gravity, 0.86 to 0.89) with or without vegetable oils for lubrication and for accelerating the solution of perfumes which otherwise would not be miscible with the paraffin oil. This problem has recently been solved by the development of a deodorized, perfumery quality olive oil which answers all purposes. Almond, apricot kernel, grapeseed, castor, avocado, turtle, and cod liver oil are all possible but rarely used ingredients.

The former heavy brilliantines which gave a shellaclike appearance to the surface of the hair are no longer popular. Light brilliantines which add a light reflecting sheen and an unexaggerated fixative action are favored. Formerly it was impossible to combine a volatile oil with a liquid paraffin base, but by using a deodorized kerosene, a fine film of oil may be deposited on the hair shaft. This also makes it possible to dilute castor oil to the proper viscosity without employing alcohol.

Petroleum jelly, hardened, tinted, and perfumed, makes what is known as solid brilliantine. The hardening is brought about by the addition of spermaceti, ceresin or ozokerite, stearin, beeswax, or one of the synthetic waxes. Because of its greasiness, this product is far less popular than the modern fixatives which may be sprayed on the hair by an atomizer. In addition to solid and liquid brilliantines, there are spirit brilliantines made of castor oil in alcohol with coloring and perfume added to make it esthetically satisfying. Less oily than the formulas discussed above, the spirit brilliantines are preferred by many because of the antiseptic action exerted by their alcohol content. These fail, however, to act very successfully as fixatives unless a damp comb is run through the hair immediately after application, precipitating the castor oil on the hair shaft where it acts as a fixative for hair that is not too thick.

Fixatives developed from a gum tragacanth base have better staying qualities and less greasiness to stain hat bands. The only difficulty is that the gum product is removed when the hair is brushed and flakes tend to accumulate on the scalp and clothing. It is claimed that if gum karaya is substituted, and alkalis are not present, flaking will not take place. Gum tragacanth's mucilaginous properties have a wide divergency. A large source of supply should be standardized and continuously checked for the viscosity of its mucilage. The gum should be dispersed by alcohol, perfume and about 10 per cent oil should be added and the water run in with constant stirring. The whiteness of the product may be improved by the addition of a little tincture of benzoin or styrax. Gum acacia may also be effective since it is a combination of the arabinates of calcium, potassium and magnesium.

Sodium alginate is a most successful base for a fixative. It is transparent and practically odorless. The viscosity may be raised by the addition of calcium ions and the lowering of the pH, due to the precipitation of calcium alginate as a jelly from the soluble alginate. All other metallic ions except those of the alkalis and magnesium act similarly.

Methyl cellulose when treated with hot water, and then cooled, forms a stable colloidal solution which is used as a fixative. Such a solution has the disadvantage of forming flakes on the hair shaft.

Other types are water-in-oil and oil-in-water emulsions. The former are supposed to contribute a better gloss and have more tenacious fixative properties than the latter. The water-in-oil type are mixtures of oil and water emulsified in the presence of a metallic soap such as calcium stearate.

Triethanolamine stearate in combination with free fatty acids forms a product with excellent emulsifying properties, practically neutral (pH at 10) and nonirritating. The fixative action is easily increased by adding a little methyl cellulose. Diglycol laurate and diglycol stearate may both be used for this purpose. The former is a pale yellow oil of low viscosity, and the latter is a white, waxy solid dispersing in water with a pH of about 6.7. Glyceryl monostearate resembles the above with a pH of 7.7. Creams having a neutral or slightly acid reaction are best for normal hair.

HAIR STRAIGHTENERS

Creams containing caustics such as sodium or barium hydroxide straighten fuzzy hair by converting the hair protein into a protein 'gel' which can be mechanically straightened and can stay so for at least a month. The process is very similar to waving hair.

At one time, the Negro people were the sole users of hair straighteners in their desire to remove the characteristic kinkiness from their hair. Today, however, beauticians recommend the process to all persons with extremely curly, spiral hair.

The problem of straightening hair may be said to be solved by the method of waving it only in reverse. Strong alkaline solutions are applied such as mixtures containing sodium hydroxide or sodium carbonate which soften the hair shaft. As it softens it is further treated with a plain or sulfonated oil emulsion or with a casein solution and gradually straightened by combing instead of being bound on spools to form the usual permanent wave. When the hairs have all been straightened they are kept so by a fixative such as a 1:1000 solution of potassium permanganate or a 10 per cent solution of formaldehyde. The mechanically straightened hair retains its new character until changed by heat, moisture or pressure. Before this effective, modern method was devised perfumed petrolatum or a gum solution of tragacanth or benzoin were used. Dermatitis sometimes resulted from the incorporated perfumes. Now, however, the strong alkalinity of the softening solution may result in dermatitis of the scalp, face and neck of the patron as well as of the hands of the operator. This cosmetic hazard will continue unless the percentage of alkalinity is controlled within limits found safe by experience. Modified types of cold wave lotions are also being experimentally employed as hair straighteners.

HAIR DYES

Hair dyes may be divided into four major classifications

- 1 Organic vegetable colorings such as henna, indigo, wood extracts including logwood, redwood, pyrogallol and tannin, and herbs including sage and camomile
- 2 Metallic preparations such as bismuth, cadmium, cobalt, copper, iron, lead, nickel, silver, and tin
- 3 Compound dyestuffs utilizing vegetable dyes mixed with any metallic preparation such as those mentioned above
- 4 Synthetic organic dyes of the amine or aniline type and sulfonated azo dyes

Of these four classifications, the last named substances are the only ones which have an immediate effect on the coloring of the hair shaft. Both vegetable and metallic substances require a series of applications before achieving the desired color and are therefore known as progressive types of dye.

Rinses

In a report from the American Medical Association's Committee on Cosmetics it was observed that the connotation of the word 'rinse' is more acceptable to the public than the word 'dye'. Nevertheless any substance which changes the color of the hair is a hair dye and not a rinse. A true rinse simply highlights certain colors in the hair.

Any mild acid is suitable as a rinse for the hair. The original purpose was to enhance the gloss of the hair by neutralizing the alkali left in the hair following a soap shampoo which gave the hair a dull lusterless appearance. Soapless shampoos do not require acid rinses. The most common preparations used in the home are lemon juice (citric acid) and vinegar (acetic acid) added to the rinse water following the shampoo.

A modern application is a combination of mild acid solutions with substances that will have hair brightening effects. Henna and camomile are commonly added to acid solutions for this purpose. The former accentuates reddish tints in brown hair, while the latter supposedly brightens fair hair. Coal tar dyes are sometimes added to give a wider range of shades. The term 'hair tint' is sometimes used interchangeably here. The same federal legislation pertains to rinses or tints as to hair dyes if paraphenylenediamine type of coloring is used.

Another recent adaptation is the shampoo rinse combination. There are several products on the market which incorporate small amounts of hair dye in a shampoo. Henna may be added only to soapless shampoos since it will dye the keratin of the hair only if soap is absent.

Products are also available which overcome the yellow discoloration which often characterizes white or gray hair. This peculiar change is more common in hair originally red or blonde. Many women apply a bluing solution to counteract this unattractive tint of the hair, since a slight blue tinge appears to the human eye as accentuating the whiteness of a substance. When certified dyes are used, these preparations should be relatively safe. The only disadvantage is that sometimes the cosmetic effect is undesirable in that too deep a shade of blue is obtained.

Bleaches

Blonde hair is maintained or acquired not by the use of a hair dye but by removing dark pigment from the hair. The bleaching agent commonly used is hydrogen peroxide. A few drops of weaker ammonia water are added to increase the oxidizing potential of hydrogen peroxide. Too large an amount of ammonia will impart an undesirable reddish shade to the hair.

So called "white henna" or "compound white henna" are misnomers since they refer to a bleaching solution which is applied in the form of a pack. There is no henna in the preparation.

Platinum blonde hair results from excessive bleaching since the degree of bleaching depends on the period of time the bleaching agent is kept in contact with the hair. The lightness is subsequently accentuated by rinsing with a certified blue color following the bleaching procedure.

The economic disadvantages of bleaching the hair parallel those of dyeing the hair. The average bleaching preparation is relatively safe to use, but continued bleaching tends to make the hair dry, brittle, resistant to permanent waving and generally unattractive and unflattering.

Vegetable Dyes

Vegetable dyes are the only ones which may be employed with the comforting assurance that they probably will not cause any dermatologic side effects. Unfortunately, however, the process is painstaking, the colors limited, and the results comparatively unsatisfactory. Our ancestors, seeking "the fountain of youth," used herbs such as camomile, sage and rhubarb either prepared at home or present in prepared hair dyes. An infusion of sage was capable of darkening light hair but only after a series of slow and oft repeated applications. Camomile kept blonde hair light without actually dyeing it, and rhubarb was used to promote golden tones in faded blonde tresses. Extracts obtained from nuts, roots, leaves, and stems of various trees were used by our grandparents. The most popular extract was prepared by boiling the green leaves or both the ripe and unripe shells of walnuts. Pyrogallic acid was thus obtained which when applied to the hair, absorbed the oxygen and produced a brown tone. Unfortunately continued use of this acid in a concentration stronger than 5 per cent is noxious.

Henna either alone or combined with indigo, is still used and has remained the only harmless dyestuff available. Colors ranging from bright red to blue black may be developed by combining *Lawsonia inermis* with the genus *Indigofera*. The dried leaves and stems of the plants are mixed with boiling water to make a paste which is applied to the hair for varying lengths of time depending on the depth or brightness of the tone desired. The resultant effect is also contingent on the original color of the hair and the strength of the paste. Unlike other dyes, simple solutions of henna and indigo do not penetrate the cuticle layer but coat it with a color which is relatively permanent. Henna alone is particularly successful when used on mousy brown hair, to which it contributes golden red highlights. It should, however, never be in contact with white or yellow hair for any length of time unless a brilliant orange is considered desirable.

Just so long as pure henna or henna and indigo compounds were used to darken or change the color of hair, no damage was evidenced, but when preliminary alkaline bleaching or permanent waving initiated pathologic changes, these compounds could no longer be considered entirely safe. Since every successful dyeing process must include bleaching to assure a uniform colorization, the alkaline agents used were bound to render the hair shaft brittle. Hydrogen peroxide, either alone or combined with ammonia, has a damaging effect. The hair at the temples becomes woolly in appearance and the shaft develops pearly nodes, at which points the hair eventually breaks, as in cases of trichorrhexis nodosa. Hydrogen peroxide with ammonia can only bleach brown and black hair successfully when it is used in a concentration so strong that it ultimately affects the hair shaft.

When pyrogallol or salts of iron and copper are added to the organic vegetable dyes in order to perfect shades of brown, henna has then to be reclassified with metallic compounds and their attendant hazards.

Metallic Hair Dyes

The metallic dyes are successful only when frequent applications are made, the desired color emerging after a series of treatments which progressively darken the hair. A dilute solution of silver, copper, iron, or lead is employed with acts on the sulfur content of the hair, depositing a metallic sulfide on the shaft. Silver nitrate, in combination with a 1 per cent pyrogallol acid solution as a reducing agent, may be adapted for all shades from light brown to black, depending on its strength. After the natural protective oils have been removed by a solution of sodium bicarbonate, the acid preparation is brushed into the hair. Then 1 to 4 Gm of silver nitrate solution combined with 20 to 30 Gm of ammonia and water are applied. When exposed to the light, or used with a developer such as pyrogallol or sodium thiosulfate, the silver is reduced and the resultant oxide of silver colors the hair. Silver salts also produce an attractive ash blonde shade for the hair but, if improperly handled, turn the scalp black. Both argyria (rare) as well as dermatitis may be concomitant features.

A great many marketed dyestuffs have lead acetate as their principal ingredient. On contact with the sulfur, a black lead sulfide is formed. Some of the formulas contain free sulfur in a solution of lead acetate to make sure that a sufficiently strong lead sulfide will be formed. During repeated applications the hair passes through a series of shades: white, yellow, brown and finally a dull, lifeless black particularly unbecoming to the face. There have been cases of systemic poisoning observed where lead dyes were in use, and such preparations should be labeled with adequate precautionary notices. Headaches, anemia, albuminuria, intestinal dysfunction, and excessive fatigue are symptoms of plumbism. Dermatitis has not been reported.

Synthetic Organic Dyes

Azo Type—This type of coal tar dye is apparently safe to use on the normal hair and scalp and is satisfactory for home use. Laboratory and clinical tests indicate that the index of sensitivity is low. However one disadvantage is that several applications are necessary to obtain the shade designated on the labeling, although

the colors do not wash out when the hair is shampooed. The Federal Government does not require a "caution" statement on the label because there is as yet no evidence that sulfonated azo dyes are harmful under conditions of use. On the other hand, the omission of the caution statement is entirely the responsibility of the manufacturer.

Amine or Aniline Type—The organic vegetable dyes and the metallic derivatives have recently been abandoned in favor of synthetic so called para dyes. These have two characteristics far superior to their predecessors. First of all, the desired color is acquired at one sitting. There is no need for cumulative applications with their unsightly intermediate shades. Second, the colors themselves, ranging from a fine auburn through all the brown tones to black, are completely natural in appearance, highlighted rather than dull, and definitely flattering to the face. A small percentage of individuals, however, are either irritated by the chemicals involved or become sensitized to these para dyes after repeated use, and develop a slight to severe contact dermatitis. Even this may be partially controlled by patch testing the dye and its oxidant in combination on the area behind the ear, before beginning each dyeing process. A Federal law and many state and municipal laws already require this as a legal prerequisite for the use of para dyes. If the skin becomes red and inflamed after forty eight hours the individual is one of the 4 per cent whom Ingram found, by testing a thousand volunteers, had a natural idiosyncrasy to the para dyes. The Food and Drug Administration reported that fifteen out of every thousand persons using para dyes will be found sensitive to the first application and twenty five others will develop a sensitivity with continued use.

In the light of more recent studies, it is problematical as to whether the preceding type of testing procedure is an adequate means of detecting sensitization to chemicals of this type. Although the problem is more comprehensively discussed under the heading of Patch Tests (page 154) it may be stated that skin tests of hair dyes should include at least three repetitive, daily applications of the dye mixture as it is employed in actual use. It is preferable to apply this mixture to the scalp margin and to extend the application into the hairy scalp as well, although other sensitive areas of the skin surface may be utilized. These uncovered applications should be made to the identical site each time. If the subject has not been previously exposed to the dye being tested the test should be repeated within ten days in order to determine whether sensitization has occurred.

Paraphenylenediamine ($\text{H}_2\text{NC}_6\text{H}_4\text{NH}_2$) is a white crystalline base, 1,4 diamino benzene, soluble in alcohol and water which requires oxidation in order to become a dye.

This synthetic dye is usually sold in two bottles: the first containing a 1 to 3 per cent aqueous solution of para; the second an oxidizer such as hydrogen peroxide or sodium persulfate or perborate. There are substitutes for paraphenylenediamine such as paratolylenediamine, para aminophenol (Rodinal) and sulfo para amino-meta cresol (Metol), which are less toxic but also less effective. Amidol may be used as a one solution self oxidizing dye. If the operator does not know whether the dye he is handling belongs to this group there are simple tests which he can use. If the bottle containing the dye is filled with a clear solution which darkens after the addition of an equal amount of hydrogen peroxide the presence of para may

be suspected: A stick immersed in dilute acetic acid will become bright red when dipped in this solution in contrast to the violet color which it assumes when in contact with the metallic dye, ferric chloride.

The untoward reactions observed on some persons are presumably due to the unstable substance, quinone diimine, which is precipitated during the process of reducing the amino benzene. Quinone diimine polymerizes into the oxidation base known as Bandrowski's base. It is only when oxidation has been incomplete that damaging toxic effects may occur. Even this risk may be successfully avoided if the hairdresser is careful to apply sufficient oxidizer to take care of all the paraphenylenediamine before the customer leaves his care. This was first noted by Scauto, who discovered that twenty eight out of forty seven subjects reacted unfavorably to the quinone compounds formed by the incomplete oxidation of these hair dyes.

The operator is also in a position to develop a contact type, eczematous dermatitis from repeated handling of these products. Rubber gloves, impervious sleeves, fatty sulfonated oils in place of soap for cleaning the hands, and the frequent application of an effective emollient cream are useful precautionary measures. Attempts to desensitize the operators by building up a gradual tolerance to the intermediate products of the paraphenylenediamine group have been unsuccessful. The sole result was an extensive eczematous dermatitis.

While the clinical features of contact dermatitis have been described at the end of this chapter, paraphenylenediamine dermatitis is characterized by the following sequence of events. A few hours after the application of the dye, mild pruritus may occur. This is followed by erythema and edema of the margin of the scalp and the face, with the appearance of papules or vesicles. In severe cases the entire face becomes so edematous that the eyes are closed and the swollen lips and oral mucosa actually prohibit speaking or swallowing. While albuminuria may be present febrile reactions seldom occur, a distinguishing point from suspected erysipelas. Within three days the edema has reached its maximum discomfort, but it may persist for as long as three weeks, with the inflammation not wholly subsiding for six weeks or longer.

In most cities the local boards of health insist on the performance of patch tests before the application of a hair dye. The technique of testing is unfortunately usually an inadequate one, as previously discussed. In addition an accurately interpreted negative reaction indicates simply that a person is not sensitive at that particular time and under those conditions. It cannot predict which persons will become sensitive at a later date although a relative margin of safety can be assured by repetition of the skin test before each application of the dye. It should be borne in mind that sensitivity can develop at any time and a negative reaction prior to the first application in no way assures a continued negative reaction. Contrary to a widespread fallacy sensitivity cannot be determined by a preliminary examination of the scalp or the hair or by merely examining the individual who is to have her hair dyed. Recent studies have also shown that the adequacy of ordinary patch testing when used alone as a measure of irritancy and allergenicity is reasonably open to question. For example a recent publication compared patch test reactions from a mild fungicide with the incidence of cutaneous reactions occurring

under conditions of actual usage of the same preparation. It was found that patch tests gave reactions which were much more frequent and severe than the incidence of dermatitis when the material was in actual use. In this study, a number of subjects who were tested by standard patch test methods showed local responses to the chemical with moderate to severe reactions. These same subjects were subsequently exposed to the daily therapeutic use of the identical chemicals without eliciting any cutaneous reactions, even after prolonged use. A possible interpretation for these findings is that in actual use the composition of the reactive chemicals may be altered by such factors as volatility, instability, and reactivity with the air, the skin and with any other materials. The substance should be rejected as an irritant or accepted as inert only when the results of patch tests are confirmed by actual use. Contrariwise exposure to a small amount of hair dye behind the ear may elicit negative reactions yet when the entire scalp is saturated with the identical chemical a severe dermatitis may ensue. Care must always be exercised when stronger chemicals are employed and the skill and experience of a careful operator will always minimize untoward side effects.

3 DERMATITIS

Clinical Features

The term contact dermatitis is used to describe an acute or chronic inflammation resulting from the external application to the skin of sensitizing (allergenic) or irritating agents. The hairy scalp is just as prone to react as the glabrous epidermis. The traumatic agents may be derived from either animal, vegetable, or chemical sources or from various interrelationships among the three. The inflammation so precipitated is characterized by redness and edema, followed by vesiculation and even at times by the development of bullae with subsequent exudation. The patient may complain of either itching or burning sensations, or alternate unhappily between the two.

The original erythema is superficial, caused by the dilatation of the peripheral vessels within the specific area defined by contact with the irritant or the sensitizer. The edema which usually becomes pronounced is intercellular and becomes the site of lesions varying in size from microscopic to macroscopic vesicles in many cases completely covering the edematous surface. Should the contact with the activating agent have been unduly long the vesicles and bullae may rupture in a few days exuding their serous contents which coagulate on exposure to the air, forming crusts.

At times the inflammation may spread beyond the original zone of contact due to absorption of the causative agent or its breakdown products and subsequent dissemination to other parts of the body. Toxic effects so produced range from simple erythema to extensive eczematous eruptions and may involve a large part of the cutaneous surface. Secondary infection is always possible and may be accompanied by fever, malaise, lymphadenopathy and impetiginization. In severe, widespread processes folliculitis, furunculosis and subcutaneous abscesses may coexist with the original dermatitis venenata.

While normally the entire cycle of the dermatitis is completed within a few days to a few weeks, chronicity results if the irritant is permitted to continue its

action. Other changes are then noted in the skin. The elastic properties of the involved scalp are lost, lichenification, induration, and hyperpigmentation ensue, with associated fissuring and crusting.

Etiology

Dermatitis venenata is dependent on several factors. The strength of the offending agent, the condition of the scalp and the presence or coexistence of hypersensitivity from previous contact. A reaction occurs quickly in a previously sensitized person. As a rule, if a person has a potentiality of becoming allergic, the latter requires an incubation period of about five to ten days. The length of time of the contact is another factor. It is customary to label a condition dermatitis venenata when more than 20 to 50 per cent of the population would react similarly to the causative factor. If hypersensitivity exists in the epidermis, it is customary to call such eruptions contact type allergic dermatitis (less than $\frac{1}{2}$ of 1 per cent of the population). The proper evaluation of a specific case depends on an investigation of the predisposition of the area involved, plus the determination of the site of the shock tissue and its reaction time, characteristic lesion, causative agent and specific test procedures.

The fine balance between the scalp's capacity to withstand external attacks and the strength of those attacks themselves may be upset by a variety of factors. Scalp resistance may be lowered by frequent washing with alkaline soaps, by hypersecretion of the sweat and sebaceous glands, and overexposure to sunlight. Congenital variations leading to ichthyotic changes and an irregularly developed keratinization of the epithelium may also play a role. Such conditions tend to render less effective the normal protection afforded by the stratum corneum. In the same way, recurrent eruptions of the scalp may lessen local resistance and lower its threshold of reactivity.

Differential Diagnosis

Should an acute dermatitis appear without warning on the scalp, the physician must immediately consider the possibility of contact dermatitis. A complete history is indicated, with special reference to the recent use of hair dyes, hair and wave lotions, cosmetic applications, and lacquers. Should a probable causative agent be discovered, the final proof often rests in the results of a patch test. However, a dermatitis may be due to an agent which shows a negative reaction on patch testing. On no account, however, should a patch test be applied during the acute stage, because it may serve to exaggerate the symptoms and produce a generalized dissemination of the dermatitis.

The types of reaction which may result from contact of the scalp with a sensitizing agent or primary irritant vary within a wide range. A well defined bullous eruption may result from a primary irritant that is, a chemical substance which, according to its strength will cause varying degrees of dermatitis in a majority of persons. On the other hand symptoms varying from a simple erythema, through edema, vesiculation and ulceration indicate a specific allergic reaction dependent in part on the sensitivity of the patient to the allergenic agent. The latter reaction develops more slowly.

Diagnosis is simplified when pruritus and/or a burning sensation follow closely upon the application of a foreign substance to the head or its appendages. This can produce any type of reaction from simple erythema to an acute inflammatory dermatitis of the scalp. An acute dermatitis spreading down along the neck and about the ears should make the physician suspicious of hair dyes or some recent chemical agent applied to the scalp. Both women and men have a tendency to deny that they use hair dye and some of them honestly believe that they never have, because of the clever advertising and promotion which allows a dye to masquerade under the pseudonym of "rinse" or "color restorer."

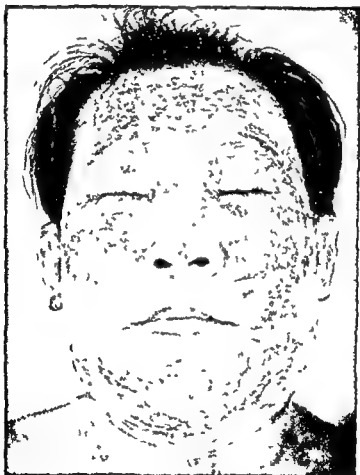


Fig. 75 —Dermatitis following use of hair dye (Courtesy Dr. F. Combes)

Dyes are not the only offenders although they are among the leading suspects. Rubefacients and similar agents used for therapeutic purposes are capable of producing an acute reaction with erythema, edema, vesiculation, and exudation, sometimes followed by pustulation due to secondary infection. Large bullae may appear on the bald areas of alopecia areata shortly after irritant applications.

Ultraviolet light used carelessly at home or in the barber shop in blistering doses may lead to pruritus, erythema, edema, and bullous formation. Overexposure to various high frequency currents can also result in dermatitis venenata.

There are many other substances which, when brought into contact with the scalp, will not cause an immediate dermatitis but will start a series of changes resulting in increased sensitivity of the scalp to that substance, so that when it is reapplied six to ten days later, it will then cause a contact dermatitis. Such agents are known as sensitizers. Some affect a large majority of humankind, others but a small percentage whose readiness to react depends on predisposing factors discussed above. For example, a statement by the Federal Food and Drug Administration concerning paraphenylenediamine indicates that out of every 1,000 persons who use this dye, 15 will be found sensitive to the first application, 25 will sooner or later acquire sensitivity with continued use of the dye, and 960 will probably be unaffected. The incubation period between the initial contact and the appearance of the dermatitis is a requisite for the development of specific, allergic hypersensitivity. In a few instances, these sensitizers may, as a result of continued usage, cause "hardening," i.e., they may lead to hyposensitivity or tolerance of the scalp to any further contact with the allergenic agent. Schwartz and Peck prepared a list of cosmetic sensitizers and primary irritants from which have been selected those which are pertinent to dermatitis of the scalp. These are listed in the accompanying table.

SUBSTANCE	USE	SENSITIZER	PRIMARY IRRITANT
Acacia	Emulsions hair preparations	X	
Alizarin	Dyes	X	
Amidol	Hair dye	X	
Ammonia	Hair wavers and bleaches		X
Ammonium bisulfite	Hair wavers and bleaches		X
Ammonium carbonate	Hair wavers and bleaches		X
Bay oil	Hair tonics	λ	
Bay rum	Hair tonics	X	
Bayberry	Hair tonics	X	
Benzoyl peroxide	Bleach	X	
Betanaphthol	Hair tonics	X	
Brazil wood	Dye	X	
Cantharides	Hair tonic	X	
Coconut oil	Shampoo	X	
Ethanolamines	Hair wavers	X	X
Ethylenediamine	Hair wavers		X
Karaya gum	Hair preparations	X	
Linseed oil	Shampoos	X	
Morpholine	Hair wavers	X	
Oxalic acid	Bleach	X	
Paraphenylenediamine	Hair dye	λ	
Potassium bisulfite	Hair waver and bleach		X
Pyrogallie acid	Hair dye	X	
Natural resins	Hair creams	X	
Resorcinol	Hair tonics	λ	
Sodium carbonate	Hair wavers	X	
Sodium persulfate	Hair wavers		X
Sodium stannite	Hair wavers		X
Zinc ammonium chloride	Hair wavers		λ

Hair lotions used in the process of permanent waving may contain organic, inorganic or synthetic products such as borates carbonates ammonium salts, phosphates, sulfites thioglycolates and aliphatic amines. Some are employed to soften

the hair so that it loses its elasticity and can take the curl. Others are used to oxidize or 'set it,' and solutions of gums or resins such as karaya, acacia, sodium alginates, gelatin, and casein complete the process by holding the curl in place. Dermatitis may develop due to the irritating effects of the reducing agents or sensitization to the resinous hair lacquers. In addition to natural colloidal material soluble in alcohol or water, there are synthetic resins such as the glycol and glycerol esters of boric acid. Schwartz reported several cases of contact dermatitis due to a strongly alkaline hair lacquer derived from maleic anhydride used as a substitute for Asiatic shellac which became difficult to import during the World War II.

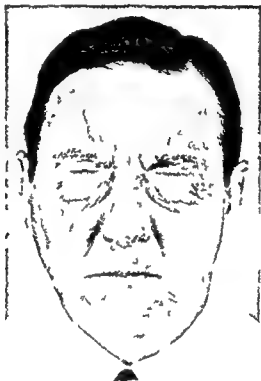


Fig. 76—Dermatitis following use of a hair lotion

In addition to dermatitis due to hair dyes, perfumes, lotions and lacquers, similar cutaneous episodes have been initiated by so called hair 'tonics' and advertised dandruff removers. High concentrations of resorcinol, betanaphthol, capsicum, cantharides and salicylic acid may act as primary skin irritants and in more dilute solution as sensitizing agents. Dermatitis contracted from these substances may either be of the dry, scaly chronic type or acutely inflammatory with varying degrees of vesiculation. Rostenberg and Sulzberger observed that in actual clinical experience hair 'tonics' produced 50 per cent more positive reactions than hair dyes.

Recently hair straighteners have had a slight vogue. Formerly the Negro people were the only ones concerned with developments in this field but now many hairdressers recommend hair straighteners to their curly headed patrons. The

applications may consist merely of perfumed petrolatum or a gum solution such as tragacanth or benzoïn (see page 142), but there is always the possibility that the compound may contain strong alkaline mixtures and contact dermatitis may ensue. Dermatitis has also been reported following contact with various types of treated leather in hat bands, hat dyes, waterproofing agents and rubber caps. Depigmentation has also been observed following exposure to substances such as agerite alba in rubber caps.



Fig. 77—Dermatitis following use of a hair straightener. (Courtesy of Dr. F. Combes.)

The scalp may also be affected by extension of dermatitis venenata from other areas (poison ivy, oak, and sumac). The active principle of *Rhus toxicodendron* is toxicodendrol, first declared a nonvolatile oil by Pfaff who claimed to have isolated it. Shelmire, forty three years later in the light of new chemical discoveries re-

characterized it as a dialyzable fraction of the okoresin soluble in water and urine. Domestic plants may also indirectly lead to dermatitis because of the increasing use of the chemical fertilizers, weed killers, and insecticides. In the same way occupational exposures to chemicals fumes and dust may cause dermatitis of the hands, face, and scalp.



Fig 78—Hatband dermatitis (Courtesy of Dr F Combes)

Patch Tests

The patch test when it is properly performed and interpreted in coordination with other factors in the diagnostic procedure is of value in establishing the cause of contact dermatitis. It should be considered in the same light as any laboratory procedure and is merely an additional piece of evidence. The Committee on Cosmetics of the American Medical Association prepared the following material which I have used as an aid in the diagnosis of dermatitis of the scalp.

Patch Test—The dermatitis in miniature and to determine what substance is the irritative agent. A small amount of substance (on a small piece of cotton or wax paper) cover with a one inch

innocuous, impermeable shield (nonwaterproof cellophane being most desirable), seal to the skin with strips of two inch adhesive tape (Patch testing kits may be purchased ready to use)

Material—For accuracy, test with original material from original container actually used. Failure to do this may cause misleading results, as some factor in that particular container and preparation may be responsible. A similar product obtained on the open market may not be identical with the one used by the patient. Since any ingredient or combination thereof may be responsible for the dermatitis patch tests should be done with each ingredient. If the specific chemical causing the dermatitis is established a preparation may be formulated for that person eliminating the sensitizer. A few companies extend the courtesy of forwarding small amounts of each ingredient in the suspected product to physicians upon request.

Site—The upper part of the body and flexor surfaces of the arms are generally more accessible, consequently these are the most popular sites. All other things being equal the most rapid reaction will occur in these areas. For maximum accuracy in comparing reactions of several substances on a group of patients all tests should be applied on the same or corresponding areas. More accurate reactions are often obtained if applied on normal skin (i.e., back of the neck, scalp margins) close to the site of the dermatitis. However, these tests should not be applied to areas where any reaction would be cosmetically undesirable, such as on the face. The patch test area to which known photosensitizing agents are to be applied should be exposed to sunlight for two hours. The time most favorable to a true reaction is during a period when the dermatitis is still present and active, since a relative hyposensitivity may develop later. However, in testing a person with a generalized dermatitis, caution and judgment must be exercised at all times.

Strength of Test Material—Concentrations should be used which will not cause primary irritation. Any standard text on allergy or dermatology will include a list of concentrations and the optimum time of exposure for the commonly used chemicals. For further accuracy effort is made to simulate conditions of natural contact therefore in testing with an antiperspirant actual perspiration may be used as a solvent.

Controls—In cases where the primary irritant action of a chemical is suspected but not definitely known controls may have to be carried out on one or more normal skins. Should a reaction occur on the control subjects on the first application, primary irritation rather than sensitizing action is indicated. In other instances to facilitate a more accurate interpretation of results a known innocuous chemical is used as a control for comparison of reactions with the suspected substance. If the solvent or suspending agent is used in applying the material for a patch test a blank or control using the solvent or suspending agent is carried out.

Pseudopositive reactions may be caused by adhesive tape or they may also occur because an area has been covered for a prolonged period. Should there be any question in this regard a control patch test utilizing distilled water can be used. A positive reaction to the control test indicates a reaction due to either of the above

two factors rather than to the suspected cosmetic. In proved cases of adhesive tape irritation, a different brand may be employed or some substitute such as Scotch tape or collodion is used.

Exposure—A positive reaction with ordinary patch test technique may appear either shortly after application or several days later. The test is rarely removed before twenty four to forty eight hours and is read each day for a few days thereafter. However, a reaction can be delayed even longer than this period of time. The patient should be instructed to notify his physician immediately if burning and itching occur at the test site any time while the patch test is in place or thereafter. The forty eight hour patch test usually detects either primary irritants or potent sensitizers. To determine the sensitizing properties of a product, The Food and Drug Administration now suggests repetitive testing for at least ten separate exposures followed by a rest period of two weeks and a final forty eight hour retest. This rather strenuous procedure is both time consuming and expensive but may serve to detect many substances previously considered of minimal sensitizing potential.

Interpretation—There are various classifications for grading reactions. However, standardization is of importance in facilitating accurate evaluation and interpretation and in rendering the patch test scientifically valuable. The following is a commonly used classification:

- 1 plus—erythema
- 2 plus—erythema edema and papule formation
- 3 plus—erythema edema and vesicle formation
- 4 plus—erythema edema vesicles and confluent bullous formation

Reactions may be classified as early or delayed. Early reactions may occur in two to four hours with vesiculation or even bullous formation. Delayed reactions may occur several days later. This indicates that a low degree of specific sensitivity is present or that a weak concentration of the sensitizer has been used. An allergic reaction sometimes tends to increase in intensity twenty four to forty eight hours after the removal of the patch whereas primary irritant reactions tend to subside with removal of the substance.

For accurate evaluation of the degree of sensitivity by means of patch tests the following facts should be included:

- 1 Concentration and amount of chemical
- 2 Sites of application
- 3 Time and number of patch test exposures
- 4 Types of reaction

Evaluation of Positive Reactions—A positive reaction does not necessarily indicate that the test substance is the cause of the dermatitis. It proves specifically that the individual is sensitive to the substance at that particular time and area of application under the conditions of the test. This indication as to the probable causative agent must be evaluated in conjunction with all other factors. The significance of the positive reaction is enhanced by a marked improvement in the derma-

titis upon the withdrawal of the test substance from the patient's environment followed by a recurrence of the dermatitis on re exposure

It is difficult to differentiate between an allergic and a primary irritant reaction from the clinical picture alone. Therefore, one must know the concentration at which a chemical will act as a primary irritant. Control tests in the case of primary irritants increase the accuracy of interpretation. A reaction which does not persist for twenty-four hours may be a false positive.

Evaluation of Negative Reactions—A negative patch test indicates that the test area is not sensitized to the agent under the conditions of the test at that particular time. A negative patch test is not conclusive evidence that the test substance is not the causative agent. Then too, there are many factors which might falsify a reaction. The test may fail to duplicate the actual mechanism which precipitated the patient's dermatitis. The patient's skin or the test site may be examined too soon and delayed reaction not considered. A contact test should not be construed as conclusively negative until it has been repeated, if possible, in an area that has been previously involved in the dermatitis.

Usage Studies—The performance of patch tests alone conveys useful information. However, when possible, they should be supplemented by observations and studies of the preparation as it is actually applied in daily use (see page 147). Some local agents may not elicit a cutaneous reaction by patch testing, yet they may still produce a dermatitis under the conditions and at the sites of constant usage. It is important to remember that most scalp preparations are applied by injunction, in many cases, vigorous massage might bring about an effect differing from that of a patch test.

Complications—In a highly sensitive individual, patch testing with a high concentration of allergen may produce cutaneous reactions which can become generalized. It is unlikely that toxic symptoms from absorption can occur since the amount of chemical used is small and the area through which absorption takes place is limited. However, on rare occasions such instances have been reported.

Medicolegal Aspects—Lawsuits and claims due to harmful effects resulting from the use of patch tests have been reported. The most certain security against such an occurrence is a well trained and qualified supervisor of the tests and adequate preliminary information on the ingredient to be tested.

Therapy

Where industrial dermatitis venenata is concerned, there are several approaches to therapy. The first is to avoid all contact with the causative agent; the second is to wear protective clothing; the third is to make a fetish of exercising due caution as to exposure to any chemicals and especially to soaps; the fourth is resorted to only if all else fails—the bowing to Fate by changing one's employment or environment to assure freedom from the irritant. Protective ointments and applications are occasionally of value provided the materials handled are not liquids in constant contact with the skin. In the latter instance the new silicone preparations may be of value and experimental studies with these agents are now in progress.

The therapy of the various types of dermatitis embraces the general principles of treatment as applied to skin disorders in general and the scalp in particular.

The details of local therapy are discussed under the heading of *Pruritus* page 401. In general the treatment of dermatitis of the scalp evolves along the lines of three cardinal principles. The first consists of the detection and elimination of the causal agent. The detection of the agent or agents may be a simple matter, and then again it may be extremely difficult and even impossible. The elimination of the substance or substances may consist merely in the cessation of the exposure, shampooing the material out of the scalp, or, in extreme cases, shaving the head. When continued usage and exposure to the agent cannot be avoided, specific and nonspecific forms of desensitization may be attempted but are infrequently successful. The second cardinal therapeutic principle consists of symptomatic relief of the dermatitis. This



Fig. 79.—Abscesses with deep cellulitis leading to folding of the scalp, an unusual complication of dermatitis of the scalp. (Courtesy McCarthy, *Diseases of the Hair*.)

may be obtained with various local applications (as described on pages 401 and 403) including wet dressings such as weak boric acid, aluminum acetate, and physiologic saline solution, and the use of bland, soothing ointments and other vehicles suitable for the scalp. When secondary infection has occurred, the pyogenic process requires care similar to that described under the heading of the various Bacterial Infections of the Scalp (page 248). Physical modalities such as x-rays (both hard rays in small doses and supersoft or Grenz rays preferably) are also indicated in certain instances. The third therapeutic principle is concerned with symptomatic relief as obtained from internal or general measures. This topic is discussed in detail on page 402 but may be stated in brief as the employment of various drugs

and other internally acting remedies to control pruritus and related symptoms. The antihistaminics, various sedatives, and similar agents are often indicated. Recently, both cortisone and ACTH have been found of great value during the acute and extremely uncomfortable stages of a dermatitis. In moderate dosage, they speedily help the patient (carefully selected and controlled) over the "hump" of the acutely uncomfortable phase of the dermatitis, at which time they should promptly be replaced by the older and less potentially harmful therapeutic measures in general usage.

This discussion of the therapeutic management of dermatitis of the scalp may seem very brief and sketchy to the reader. However, inasmuch as the therapy of dermatitis and its complications actually occupies a major position in the management of most scalp disorders, it was considered preferable to avoid repetition and merely to generalize at this point in the text. Page references are listed and refer to specific components of the therapeutic armamentarium adequately described elsewhere in this volume.

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CHAPTER III

ALOPECIA

The subject of alopecia embraces a major portion of this text. The individual disorders are discussed elsewhere under specific classifications of the various scalp diseases. The following classification has been found suitable.

- 1 Congenital alopecia and congenital anomalies associated with alopecia
- 2 Acquired alopecia
 - A Cicatricial alopecia
 - 1 Local
 - a Physical agents: mechanical cold, heat actinic - electric x ray radium atomic bomb
 - b Chemical agents
 - c Bacterial infection: impetigo contagiosa impetigo of Bockhart furunculosis carbunculosis lupoid sycosis folliculitis keloidalis dissecting cellulitis of the scalp
 - d Fungus infections
 - e Virus infections
 - f Acneiform disorders
 - g Miscellaneous: pseudopelade folliculitis decalvans follicular lichen planus severe seborrheic dermatitis
 - 2 Local and/or systemic
 - a Lupus erythematosus
 - b Lupus vulgaris
 - c Syphilis
 - d Tuberculosis
 - e Leprosy
 - f Scleroderma
 - B Noncicatricial alopecia
 - 1 Local
 - a Male pattern alopecia
 - b Neurodermatoses (scalp disorders with psychogenic components)
 - c Seborrheic diatheses
 - d Trauma
 - e Fungus infections
 - f Bacterial and other simple pustular infections
 - g Virus infections
 - h Drugs
 - 2 Local and/or systemic
 - a High fevers and infectious disease
 - b Syphilis
 - c Exfoliative dermatitis
 - d Endocrine dysfunction
 - e Nutritional disturbances
 - C Cicatricial and/or noncicatricial alopecia

In this chapter discussion has been limited to congenital alopecia and anomalies male pattern alopecia cicatricial alopecia due to physical agents medicolegal aspects of hair morphology, pseudopelade, and folliculitis decalvans

1 CONGENITAL ALOPECIA

Clinical Features

The infant or child who either has been born without hair of any kind or whose scalp has produced lanugo hair which is never replaced by terminal hair may be characterized as suffering from congenital alopecia. In many instances it is possible genetically to relate this symptom to other members of the family and/or his progenitors. Eyebrows and eyelashes are lacking and the lanugo hair rarely grows beyond an inch in length. Other portions of the body which would normally exhibit hair at various stages of the patient's endocrine development remain hairless throughout life.



Fig 111—Hypotrichotic congenital ectodermal defects sister and brother. (Courtesy Sutton and Sutton. *Handbook of Diseases of the Skin*. Original photograph by Dr O G Costa.)

The cases discussed in medical history fall naturally into two groupings: those whose alopecia is the sole deviation from the norm and those whose alopecia is associated with other ectodermal defects. One often finds dental aplasia characterized by widely spaced missing striated or undeveloped teeth with an abnormal tendency to early decay in the patient suffering from congenital hypotrichosis. Dystrophic nails and hypofunction of the sweat glands are often concomitant features.

In congenital alopecia the question sometimes arises as to the period of development during which the abnormalities occur or first make their appearance. The classification of Bonnet is adequate for subdivision of these cases.

- 1 Congenital absence of hair associated with anomalies of the nails and teeth is indicative of the fact that developmental ectodermal defects began early in intra uterine life.

- 2 Congenital absence of hair in individuals with normal nails and teeth indicate that the ectodermal abnormalities occurred late in intra-uterine life
- 3 Congenital absence of hair found in otherwise normal subjects and present until the time of puberty (when the endocrine changes result in a normal growth of hair) simply indicate a delayed ectodermal development

There have been various classifications of the types of congenital alopecia Hyde studied this question thoroughly and formulated several groupings which were modified as follows by Ormsby and Montgomery

- 1 Intra uterine atrichia—Complete universal absence of all forms of hair at birth with no tendency to grow hair in later life This intra uterine atrichia is believed due to a failure of development of the hair follicle and is the rarest form of congenital alopecia
- 2 *Universal hypotrichosis in which hairs develop in all parts of the body, but later fail to be replaced by normal adult hair*
 - a Infant is born with the relatively long hair of most normal infants, which in due time falls out and is replaced by lanugo hair which does not develop at some later period into the normal hair of the adult
 - b The infant is born with lanugo hair, which may persist indefinitely as such, instead of being replaced by normal adult hair, or may be replaced by a scanty or poorly formed and patchy type of adult hair
- 3 Complete or partial absence of hair at birth in definitely circumscribed areas, such as the scalp, eyebrows, and to a lesser extent, the axillary, inguinal and bearded regions In the latter three instances, failure of
- 4 birth : There have also been several reports of congenital alopecia due to psychic and traumatic types of alopecia areata developing during pregnancy
- 5 Universal or circumscribed hypotrichosis in association with other ectodermal defects, as evidenced by dystrophic changes of the teeth, nails and skin

Several other classifications of congenital alopecia have been advanced Most of these groupings are not as complete as the preceding paragraph One of these classifications includes several minor variations some of which are more aptly described elsewhere in this volume This classification of five varieties of congenital alopecia was originally suggested by Dubreuilh and Petges

- 1 Congenital alopecia of a circumscribed type in which plaques of baldness develop as the result of the presence of new usually of the papillomatous variety Pigmented and papillomatous new are often found elsewhere on the body surface
- 2 Universal alopecia with the exception of a few tufts of lanugo hairs in the vicinity of the posterior fontanel or the median line
- 3 Circumscribed alopecia occurring in the frontal and fronto parietal regions, supposedly due to the result of pressure from obstetrical forceps

- 4 Circumscribed alopecias along the cranial suture lines considered due to enlargement of the cranial vault before union of the fontanel's occurs
- 5 Circumscribed or diffuse alopecia without apparent cause At puberty these areas become covered with hair

This classification is obviously an inadequate one, although it serves to describe localized patches of alopecia in association with nevus and various types of congenital alopecia resulting from arrested development of the skin

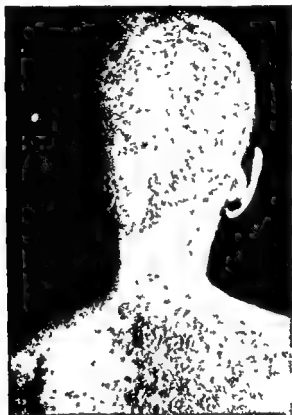


Fig 11—Congenital alopecia syndrome (Courtesy Sutton and Sutton, Diseases of the Skin Original photograph by Dr H Swetzer)

There are individual deviations in each case—a 13 year old girl who was totally hairless except for down on her cheeks and forearms, and was, in every respect other than this, a healthy and normal appearing child grew a tuft of hair (which subsequently disappeared) in the occipital region during her menstrual periods. A woman, her son, daughter and grandchild all suffered from generalized alopecia since birth, except for a few scattered and isolated terminal hairs on their scalps and in the region of the eyebrows.

This condition is not so rare when found in relation to other abnormalities. Twenty two members of a family of sixty six exhibited simultaneous dystrophy of hair and nails and similar symptoms have been traced through several generations.

of one family. Upshaw and Montgomery carefully described what they preferred to call *hereditary anhidrotic ectodermal dysplasia* and discussed the differential diagnosis in detail. In these cases they found a generalized alopecia, usually subtotal, a soft, dry feminine type of epidermis, infantile breasts, decreased, absent, or deformed teeth, pubic hair characterized by the inverted pyramid, frontal bosses, depressed nasal bridges and acroecrosis with a distinct intolerance to heat. All of these phenomena are evidenced in the first year of the patient's life.

Hidrotic ectodermal dysplasia differs from the above, not in the appearance of the congenital alopecia, but in the fact that the sweat glands are normal as is the facies, that the sebaceous glands function subnormally, that the fingernails have a short, thick, elevated tip, and that there is a hyperkeratosis of the palms and soles.

In addition to the generalized alopecias discussed above, Sabouraud reported a few cases of *congenital triangular alopecia*. The child is born with a triangular area of hairless scalp just inside the anterior hair border where the frontal and temporal portions meet. It measures 3 to 4 cm. on its largest side, and its base is directed toward the forehead. The hypotrichotic skin is completely normal.

Most textbooks list the designation '*agenesia pilaris* (Jacquet)' as a connecting link between alopecia of a congenital type and alopecia areata. According to McCarthy, this is a rare condition in which an infant is born without hair on the scalp, eyelash or eyebrow regions. The scalp is covered with a fine powdery desquamation with occasional lanugo hairs scattered over its surface. In contrast, the infant with monilethrix is born with a normal amount of hair which is lost six to eight weeks after birth. In this condition (*agenesia pilaris*), one type is described as occurring before puberty, and the hairs of the outer half of the eyebrows and back of the neck are almost completely lacking. The other type occurs during puberty and involves the regions where hair normally develops at this time. The mustache remains in the lanugo stage and the bearded region discloses only patchy tufts of hair widely separated from one another. The hair on the scalp is usually very fine and short. These cases should not be confused with atrophy of the hair due to ichthyosis of the scalp occurring as part of a general ichthyosis. The ichthyotic child has a normal amount of hair until the fifth week post partum at which time a generalized ichthyosis appears and gradually increases in intensity until the scalp is covered with a thick mass of scales. This thick scalp cap gradually chokes off and atrophies the hair almost to the point of complete disappearance.

Although such cases have been described as *agenesia pilaris* in the majority of textbooks, I do not believe they are entitled to a separate designation. According to Sabouraud, all his cases were due to congenital syphilis. In most instances careful examination discloses syphilis to be the causative factor, and *agenesia pilaris* then becomes a practically nonexistent entity. In those few cases where an etiologic agent cannot be determined, it is more aptly classified under the heading of one of the less common forms of congenital alopecia.

Pathology

Microscopic studies indicate that in congenital types of alopecia the normal hair follicle was replaced by strands of epithelial cells or cysts, thought to be degen-

erations of rudimentary sebaceous glands. The healthy sebaceous glands exhibited involuted epithelial tubules at their bases. Some were solid, some cystic, but all lacked any communication with the outer surface of the skin.

Proliferating masses of epithelial cells indicating suppressed hair follicles were sometimes found deep in the connective tissue in scattered groups. In certain cases hair papillae have been found without the follicle. Muscles, glands, nerves, and blood supply appeared to be normal.

It is thought that when these structural abnormalities began in early fetal existence, the anhidrotic ectodermal dysplasia resulted, but when these defects developed late, only congenital absence of hair was present in an otherwise normal body.

Etiology

There have been many discussions about the etiology of congenital alopecia. Early writers were inclined to suspect a syphilitic causation because of the facial similarity between anhidrotic patients and those with congenital syphilis. This, however, is obviously incorrect since the concomitant symptoms are lacking in the syphilitic and in those cases of congenital alopecia in otherwise perfectly normal patients it would have no bearing whatsoever.

Since follicular atrophy during intrauterine life is an obvious part of its history, certain mechanical causes have been suggested which might have prevented normal follicular development, such as a long continued pressure of the fetal head against the symphysis pubis with subsequent destruction of the incipient hair roots. Even high forceps at birth have been mentioned as a causative factor, particularly in cases of triangular congenital alopecia, but this certainly could never become an hereditary syndrome.

The syndrome has also been discussed on the basis of endocrine dysfunction, an hypothesis which since proved undemonstrable. It now appears that the sole etiologic concept which is wholly defensible is that of heredity. First it was classified as a recessive and sex limited hereditary disease of males, but as more and more cases were reported it became obvious that the method of genetic transmission is not identical in all patients. Modern geneticists now claim that one peculiarity of human genetics is that the same abnormal character may be sex linked, recessive or dominant in different families. Each of these modes of inheritance has already been illustrated in the study of genetic transmission of the complete anhidrotic syndrome.

Therapy

The etiology of this syndrome and all practical experience with its treatment leads us to conclude that therapy is useless unless specific endocrine factors or systemic disease processes are causative and can be influenced therapeutically. However, a prognosis should be advanced with caution inasmuch as the occasional patient with congenital alopecia may exhibit surprising changes at the various periods of endocrine change. In general, however, the only remedy for congenital alopecia is usually offered by the wigmaker, not the physician.

2 CONGENITAL ANOMALIES ASSOCIATED WITH ALOPECIA

Several anomalous conditions present at birth often show an associated circumscribed or generalized loss of hair

CONGENITAL ECTODERMAL DEFECT

This condition is characterized by the incomplete development or absence of the epidermis and its appendages from certain areas of the body. The skin is dry and smooth and lanugo hair is absent from the major portion of its surface. The hair is scanty and thin on the scalp as well as on all the usual hairy parts. The nasal bridge is depressed as in the saddle nose of congenital syphilis. The teeth are defective both numerically and morphologically. The nails often show dystrophic changes and congenital cataracts have been reported. A subnormal mentality is not an uncommon component of this picture.

Congenital ectodermal defects usually occur in males and are familial. The cause is unknown. There is a complete absence of sweat glands and often of the sebaceous glands as well. The arrest in development occurs early in intrauterine life. Therapy is of little value although avoidance of heat and physical exertion renders the patient more comfortable.

CONGENITAL DEFECTS OF THE SCALP

Cicatricial lesions occurring in the scalp of newborn infants were described in detail by Anderson and Novy. They classified these lesions as congenital defects and localized them to the vertex, the sagittal suture and over the parietal bones. Cases previously described as congenital atrophy belong in this group. The lesions vary in size from 1 mm. to 5 cm. The form of the defect is usually circular but it may be longitudinal or irregular. The area of involvement is hairless at birth but some hair may appear in a few weeks. In other cases the area may remain bald permanently. The lesions are sharply circumscribed, depressed and atrophic or scarlike patches which at times present punched out margins. The wound itself may appear fresh at birth and is covered with coagulum or granulation tissue. If seen when the child is older a round atrophic scar is present. The atrophy may be superficial or deep, occupying the entire skin and subcutaneous tissue. Other congenital malformations may be present. As a rule the defects are single although in occasional instances they may be multiple. These lesions should not be confused with circumscribed scleroderma of the linear type referred to as *en coup de sabre* (page 467). Histologically there is total absence of sebaceous and sweat glands and there are other evidences of atrophy.

Several hypotheses have been advanced to explain the formation of these lesions. The first is that they are due to amniotic adhesions. According to this theory abnormalities occur in the early amnion and there is a persistence of adhesions between the amnion and the fetal skin. As accumulation of the amniotic fluid increases bands (Simonart's bands) are formed. When sufficient liquor amnii has been formed such bands are torn loose from the skin of the fetus and a cutaneous defect remains. Some authors believe that a careful search of the placenta and membranes would reveal such amniotic bands in an increasing percentage of such

cases. Others feel that even the integrity of the amnion does not speak against an amniotic origin. Sitzenfrey stated the belief that the amniotic origin of such defects can be demonstrated by histologic methods. Because the sweat and sebaceous glands together with the hair buds are formed about the fourth month of fetal life, it has been assumed by some that such amniotic fetal adhesions must be present before that time. This is because the accessory cutaneous glands are always absent in cases exhibiting the congenital defect.

The other theory of causation is that advanced by Greig. He stated the belief that such defects are due to arrested development. Ingalls in an excellent article defended Greig's point of view that the cause of this anomaly is due to arrested development and not to amniotic adhesions. He pointed out that in the human embryo the dorsal midline particularly the region of the head is subjected to extremely important rearrangements. It is only natural then to expect anomalies in this area. He then mentioned extreme anomalies such as anencephaly and rachischisis and wondered why minor defects such as cutaneous changes are so rare. He reported the examination of several embryos in which the only defect was that of bullae forming just beneath the epidermis. If the epidermis were torn away or if the bullae ruptured in cases of this kind scarring would occur and the clinical features of a congenital defect of the scalp would result. He further stated that while the fetus is surrounded with amniotic fluid such lesions show little tendency toward healing but this does take place rapidly after birth. He has never noted any amniotic adhesions.

There is no treatment that has any effect on the lesions. Anderson and Novy refer to a mortality rate of 20 per cent the cause of death being meningitis.

CHONDRODYSTROPHIA CALCIFICANS CONGENITA (WITH PSEUDOPELADF)

Pseudopelade and follicular atrophoderma in combination with skeletal deformities such as have been described as end results of chondrodystrophia calcificans congenita were observed by Curth in one member of one and in several members of another family. In addition other congenital defects such as incontinentia pigmenti, cataracts and polydactylia were seen in some members of the family. Curth states that this syndrome is caused by inherited hypoplastic changes of the fetal cartilage and skin. Great variation in the degree of bone involvement has been observed in the various cases. Lesions resembling eczema are present at birth on body and scalp. These as well as the disturbance of the cartilage, undergo various postnatal stages. The syndrome has been observed only in females. Although the scalp lesions have been described as pseudopelade they are more aptly classified as cicatricial alopecia.

CONGENITAL SKULL DEPRESSIONS

Congenital depressions of the skull usually involve the parietal bones. Spoon shaped depressions are present and there is no loss of hair. They gradually disappear about the third or fourth year of life. The usual cause is pressure on the cranial bones by the sacral promontory.



Fig 87—Congenital Calcific Alopecia. A Cicatricial alopecia. B Right lower extremity showing follicular atrophoderma. C Biopsy from region of follicular atrophoderma showing hyperkeratosis, loss of rete pegs, and perivascular infiltrate in cutis. (Courtesy Dr. H. Curth and J. Invest. Dermat.)

CONGENITAL AURICULAR FISTULA

These lesions are often mistaken for furuncles or infected sebaceous cysts. Clinically they appear as shallow depressions in the skin or as fistulous openings in the preauricular region. Montgomery states that the sinus tract may be superficial and run immediately beneath the skin or extend deeply into the soft parts and parallel the external auditory canal. It may lead to the middle ear. As a rule there is merely dimpling or a short fistulous tract without dimpling. The lesions may be unilateral or bilateral and there may be two or more on one side. They may or may not intercommunicate and the tracts may or may not secrete depending on the presence of sebaceous and sweat glands. Not infrequently the tract becomes blocked and cysts of varying size develop. If these cysts are secondarily infected the lesion becomes swollen, red and painful. Occasionally a granulomatous appearance is presented suggesting scrofuloderma.



Fig. 83—Chondrodystrophia calcificans congenita. A Congenital alopecia. The hair is blond, dry and lusterless. B Incontinent pigmentation. (Courtesy Dr. H. Curth and J. Invest. Dermat.)

These lesions may be due to incomplete closure of the first branchial pouch or to incomplete fusion of the embryonic tubercles which form the auricle. They are often misdiagnosed and treated with ineffectual local measures and antibiotic therapy. The latter will control the secondary infection but recurrence can only be prevented by thorough surgical excision of the entire fistulous tract. This procedure should not be minimized inasmuch as the tracts are often deep and tortuous and require careful dissection.

PACHYONYCHIA CONGENITA

This rare congenital anomaly was originally described in males by Jadassohn and Lewandowsky. It is characterized by dystrophic changes in the nails, palmar and plantar hyperkeratosis, anomalies of the hair, leukoplakia, follicular keratoses of the acneiform type, particularly about the knees and elbows, and dyskeratosis of the cornea. The scalp may show any of the changes described under the various types of congenital alopecia.



Fig. 84—Congenital auricular fistula with granulated changes. (Courtesy Dr. F. Coates.)

WERNER'S SYNDROME

This rare hereditary disease is characterized by normal birth and development until adolescence, undersized stature, gray hair, male pattern alopecia, scleroderma and poikiloderma, trophic ulcers of the legs, juvenile cataracts, hypogonadism, diabetes, calcification of blood vessels, osteoporosis, and metastatic calcifications. These patients present some of the cutaneous features observed in the disorder known as congenital ectodermal defect, also a type of congenital ectodermal dysplasia.

3. MALE PATTERN ALOPECIA (COMMON BALDNESS)*

Clinical Features

The common type of alopecia of the patterned variety is responsible for more than 90 per cent of all cases of baldness. Although occasionally described as idiopathic premature alopecia, presenile alopecia, senile alopecia, and the like, these terms are merely descriptive of various phases of male pattern alopecia and should be discarded.

Hamilton described the usual appearance of the scalp from prenatal life until old age. In the youngest fetuses (lengths of 141 to 144 mm from crown to rump), hair was not present. When the crown to rump length exceeded 158 mm, hairs were present on the surface of the scalp. The first hairs to appear on the surface of the scalp are very fine in texture. This hairy coat extends without interruption to cover the forehead. In progressively older fetuses, the hairs of the scalp become more coarse. In fetuses which were considered to have reached term, the hair over the frontoparietal and frontal regions, but not over the tonsure, was thinner than elsewhere on the scalp. Soon after birth a marked alopecia develops in many babies in the same areas that become bald in adult men, that is, in the frontoparietal and frontal areas, and even over the tonsure. This process continues for weeks or months and occurs in both sexes. While hairs are still being lost, new hairs appear on the surface of the scalp. Then, by the end of the first or second year, most children have a completely hairy scalp. This continues until adolescence, at which time some amount of hair is lost along the anterior border of the scalp in most members of both sexes. The loss of hair tends to be more pronounced in boys than in girls. From puberty on, this was observed to become increasingly common with advancing age in males until the seventh decade, when the frequency was highest. In females, the incidence did not increase after the fifth decade. Hamilton noted that in the sampling of an average group, advanced stages of baldness were not found in any woman although they were present in 58 per cent of men above the age of fifty. In Hamilton's group of 526 subjects between 20 and 92 years of age, no type of alopecia other than common baldness was observed. From this he concluded that in the general population the common forms of baldness account for almost all instances of alopecia. He also observed that in scalps in which there was a lack of recession of the anterior regions, normally characteristic of children until adolescence, no alopecia developed in 95 per cent of twenty men who failed to mature sexually. This type of scalp was eliminated after androgenic treatment in all of eleven sexually immature men and was present in only two of twenty four virilized women. After normal sexual maturity, this type of scalp (i.e., those which lack recessions of the anterior regions) is retained in only 4 per cent of men but in 21 per cent of women. A study of pedigrees in normal men showed that a tendency toward this type of scalp was inherited by certain Caucasian families and was a racial trait in Chinese. Alopecia was less common and, when present, occurred at a later age in Chinese men than in Caucasian men.

*The greater part of this material has been obtained from the studies and publications of Dr. James B. Hamilton with whose kind permission it is here presented. I also wish to express my appreciation to Dr. Roy W. Mincer, the editor of the *Annals of the New York Academy of Sciences* for his cooperation.

The scalp hair shows changes of a fairly typical nature with advancing age. Each successive generation of hair lessens in diameter, the hirsute growth becoming finer and finer until the follicle no longer produces the hair, progressive immaturity finally ending in atrophy. The alopecia is never diffused; it has a specific locale and follows a well known course until it achieves the typical patterns of male baldness. Although the hair becomes thinner in the marginal area, it is never completely lost, and extreme examples of hypocratic or classical baldness will still maintain a border fringe a few centimeters wide festooned around the back of the head from ear to ear.

Hamilton has recently classified the various patterns of ordinary baldness. The following types of scalp categorize the typical sequences in the development of common baldness by Caucasians. There are, of course, variant patterns and slight modifications of these types of scalp, so that no single sequence is followed uniformly by all subjects.

Scalps Which Are Not Bald Types I to III—*Type I*—The essential feature of Type I is the absence of bilateral recessions along the anterior border of the hair line in the frontoparietal regions. In this and in the other scalp types there is a variant form in which the entire anterior border of the hairline lies high on the forehead.

Type II—The anterior border of the hairline in the frontoparietal regions has triangular areas of recession which tend to be symmetrical. These areas of denudation extend no farther posteriorly than a point 3 cm. anterior to a line drawn in a coronal plane between the external auditory meatuses. Hair is also lost or sparse along the midfrontal border of the scalp, but the depth of the affected area is much less than in the frontoparietal regions. A region of midfrontal sparseness or denudation of such depth that it extends as a band of uniform width along the entire anterior border of the scalp is so rare that no Type IIA was employed in routine classification.

Type III—To avoid inclusion of disputable instances of baldness, borderline cases are listed separately as Type III. Also included in Type III are scalps in which classification is rendered inaccurate due to scars, lateral asymmetry in denudation, unusual types of sparseness and thinning of the hair, and other factors. In the present study, less than 1 per cent of the scalps were placed in Type III. Stated in reverse, more than 99 per cent of the scalps could be classified as Types I, II, IV, V, VI, VII, or VIII.

Scalps Which Are Bald Types IV to VIII—Decision as to the minimal extent of denudation sufficient to constitute baldness was a wholly arbitrary one that was made after a study of scalps in 200 subjects.

Type IV—This represents the minimal extent of hair loss considered sufficient to represent baldness. Most Type IV scalps have deep frontotemporal recessions which are usually symmetrical and are either bare or very sparsely covered by hair. These recessions extend farther posteriorly than a point which lies 3 cm. anterior to a coronal line drawn between the external auditory meatuses. In most subjects some hair is also lost along the midfrontal border of the scalp. If hair is sparse or lacking as a broad band along the entire anterior border of the hairline, the scalp is classified as Type IVA.

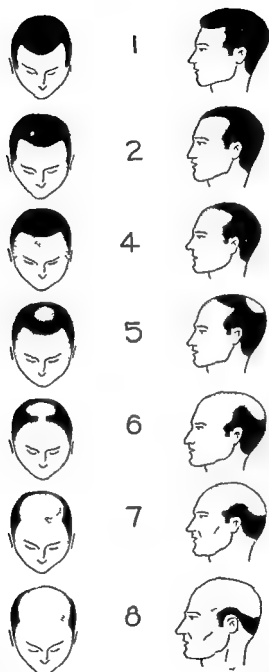


Fig 11—Sketches of the categories of scalp baldness which were set up for classification and grading of the extent of common baldness. The types of scalp are described in detail in the text. Type 3 is not included because of the variety of conditions included under that classification. (Sketches and text based on material of Dr James H Hamilton)

In old persons of both sexes but particularly in men the minimal amount of sparseness or loss of hair that qualifies as baldness may occur chiefly on the crown. If denudation on the forepart of the head is not more extensive than in Type IV these scalps with absent or sparse tonsural hairs are classified as Type IV (old).

Type I'—With advancing age men tend to acquire not only extensive fronto-parietal and frontal recessions but also a sparseness or absence of hair on the crown. Such extensive but localized areas of denudation on both anterior and postero-superior portions of the scalp are classified as Type V.

Types VI, VII, and VIII—These types are characterized by areas of alopecia which have the outline of a horseshoe when the head is examined from above. In Type VI the tonsural region of alopecia remains separated from more anteriorly located areas of denudation by a laterally directed bar of scalp in which the hair is only slightly sparse. This bar bridges the region between the fully haired areas on each side of the head. A peninsula or an island of hair lies in the midline anterior to this laterally directed hairy bridge. In the variant pattern Type VIA the peninsula or island of midfrontal hair is sparse or lost.

In Types VII and VIII the horseshoe shaped area of sparse hair or of denudation is unbroken by any well haired laterally directed bridge of scalp. This is a result of the spread and confluence of the tonsural and the more anteriorly located regions of alopecia. The bare area also extends farther laterally and posteriorly than in Type V.

In Type VIII large hairs within the horseshoe shaped area of alopecia occur only as isolated structures and not in clumps of 100 or more terminal hairs. In most Type VIII scalps the horseshoe shaped area is still indented symmetrically on its lateral aspect along a coronal line connecting the external auditory meatuses.

In the most advanced cases of baldness all clumps of hair may be lost within the horseshoe shaped area of alopecia. The regions of denudation spread still farther peripherally both laterally and posteriorly. The hair may also become sparse and thin on the back of the neck in a semicircle around the ear and on the antero-lateral aspect of the head.

Hamilton also states that scalps of Type I in which frontoparietal recessions are absent is one of the most interesting patterns of scalp hairness. This pilary state characterizes most children from the first or second year of life until adolescence and is retained in the majority of men who fail to mature sexually. Type I scalps were lost by all of 11 sexually immature men who were treated for long periods of time with androgens. Type I scalps were present in only 2 of 24 virilized women whom Hamilton had the opportunity to study.

In normal individuals the disappearance of Type I scalps follows sexual maturation. Maturation is somewhat less likely to abolish a Type I scalp in females than in males. Between 20 and 92 years of age, Type I scalps were present in 21 per cent of the normal women and in 4 per cent of the normal men. At all ages after puberty Type I scalps were more common in females than in males.

Type I scalps are retained in some men who have matured sexually and have what appears to be quite normal testicular function thereafter as judged from physical examination demonstrated fertility and their titers of urinary androgens.

and ketosteroids. Study of the pedigrees of these men indicates that Type I scalps in normal Caucasian men represent familial traits. In normal Caucasian men with Type I scalps, a similar pilary status is much more common in their adult male relatives than among men in the general population.

A tendency to retention of Type I scalps in men seems also to be inherited as a racial trait among Chinese and full blooded Indians, in whom the condition is more common than among Caucasian men. In fact, Type I scalps were found as commonly in Chinese men as in Caucasian women. Quite apparently, then, the capacity for slight degrees of denudation of the scalp in children of Oriental and Caucasian races depends upon a genetic predisposition, just as in the case of susceptibility to development of an extensively bald scalp.

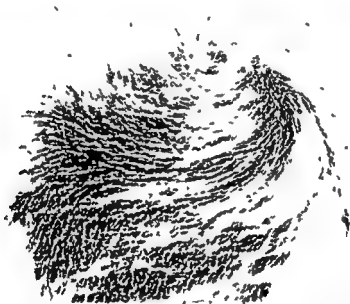


Fig 86—Diffuse alopecia in a woman. This type of alopecia is actually male pattern baldness occurring in the female sex, and is of more frequent occurrence than is generally believed.

Almost all Caucasians of both sexes who do not retain Type I scalps have acquired at least a Type II scalp within a decade after puberty. Type II scalps are the most advanced stage of denudation acquired by most women but are only an intermediate stage in most men. In men, Type IV is, like Type II, an intermediate stage, and, in most men, it is replaced by more advanced baldness by the seventh decade of life. In women, Type IV represents the most advanced stage of alopecia, and the incidence of this type of baldness was found not to increase after the fifth decade of life. More advanced stages of baldness, which form a horseshoe shaped area and may be referred to collectively as Types V to VIII, increase in incidence with age in males until at least the seventh decade.

In my opinion, Type IV patterns of alopecia do not represent the most advanced stages of patterned baldness in women. I have examined many women with patterned loss of hair who do not fall specifically into any of the above groupings. These women show a slight degree of frontoparietal recession, but their major hair loss commences approximately 1 to 3 cm posterior to the anterior scalp margin and diffusely involves the entire mid-portion of the scalp. Although complete loss of hair does not occur in these women, the hair often becomes quite sparse. Careful examination of their pedigrees usually reveals that a similar diffuse alopecia was present in their mother's and maternal grandmother's scalps. The hair loss is invariably increased subsequent to each pregnancy due to the reasons mentioned in the section on endocrine physiology (page 101). I have named this condition female pattern alopecia.

The rate of hair loss is more or less individual. In the early stages of male pattern alopecia, many hundreds of hairs may be shed in one day. After several months this may decrease to approximately thirty. Because of these intervals of remission, advanced baldness is seldom noticed before the man is 30 years old even though the original losses may have taken place shortly after puberty. It was Sabouraud's opinion that if half the hair were not lost by thirty, the patient would never suffer from exaggerated alopecia. In the majority of instances, this observation is questionably prophetic. During the periods exempt from profuse hair fall there is the usual growth of new hair but never in sufficient quantity or quality to make up for the previous loss, since these fine, silky hairs are after all, being produced by papillae already undergoing progressive atrophy. Their eventual occurrence is glibly prophesied by the quack "hair grower," and their actual appearance is attended by great fanfare and mutual congratulatory exchanges. Unfortunately, they rarely eventuate into normal, mature hair.

Pathology

Few skin changes are apparent in histologic sections from scalps showing male pattern alopecia. The hair follicles are atrophic in the affected regions and there is an increased connective tissue infiltration of the subcutaneous fat. The sweat and sebaceous glands show little or no change.

Etiology

Numerous theories have been advanced to explain ordinary male baldness or, as it is preferentially termed, male pattern alopecia. The literature dealing with this subject is voluminous and I have attempted to classify and digest the most recent studies and deductions as to its nature. At the present time, most scientific observers agree that common baldness is due primarily to genetic, hormonal, and aging factors although local and systemic disease may play a minor and usually incidental role.

A recent study by Szasz and Robertson carefully reviewed the various theories advanced with respect to the pathogenesis of male pattern alopecia. They observed that Schein was one of the first to suggest that scalp tension was the crucial etiologic factor in the production of ordinary human baldness. He expressed the belief that contraction of the epicranial muscles reduced the circulation of blood and lymph by compressing the skin and connective tissues overlying the galea aponeurotica onto

the galea. He proposed that it is not alopecia per se which is inherited but the shape of the skull favoring its development, and concluded that the nourishment of skin and hair is the determining factor in the etiology of baldness. He stated that the difference between men and women in relation to alopecia was due to a differential development of the epicranial muscles. Just as the general bodily musculature is more highly developed in men, so is the musculature of the epicranium. Schein stated further that the heads of men are generally larger and more convex on top than those of women, these factors favor the development of greater tension over the top of the head in men.

In another paper published a few months later, Schein referred to the work of Pohl-Pincus, who many years earlier (1875) had come to the same conclusions by a different approach. Whereas Schein relied on anatomic and mechanical considerations, Pohl-Pincus had approached the problem from a histologic standpoint. Most of Schein's second paper consists of quotations from the early work of Pohl-Pincus, who found a tight compression of tissue spaces in the scalps of bald men and attributed the loss of hair to this. This was in contrast to the finding that the scalp tissues over the galea in women appear loose on histologic examination. The looseness of the connective tissues of women was compared by Pohl-Pincus to that characteristic of children.

Szasz and Robertson state that although he was apparently unaware of the early work of Pohl-Pincus and Schein, Young reported some interesting studies concerning this problem in the course of which he came to essentially the same conclusions as did the two authors cited previously. In his first paper, Young stated that the "constant pattern of senile alopecia suggests that there is a definite anatomical basis for this condition. Injection studies reveal a richer vascular supply in the marginal (muscular) zone of the scalp than in the central (aponeurotic) zone." Alopecia, as is well known, is confined to the central areas, whereas hair always persists in the marginal area overlying the occipital and auricular muscles. Further, Young stated that "the bald area shows a loss of hair follicles and fat with increased fibrosis in the subcutaneous layer." Finally, he summed up his experiments as follows: "Tension areas were produced experimentally in rhesus monkeys by suturing the free scalp edges after excising elliptical segments, thus reducing the circumference. These operations resulted in persistent baldness closely resembling the human types."

In his second paper, Young showed that scalp tension leads to reduction in the thickness of the soft tissues and interference with the circulation, with consequent loss of hair. He wrote: "X-ray studies of a series show that normally the soft tissue of the scalp is almost twice as thick as that over the bare forehead. This difference is apparently due to the greater amount of fat in the scalp and each globule is well vascularized. In the bald person this differential is lost and the scalp thickness is reduced to that of the forehead giving a thin halo over the cranium. All the bald heads showed this thinning by X-ray studies and our autopsy cases showed loss of fat beneath the bald areas." Finally, he concluded that the cause of baldness is due to a disproportionate growth of the brain and of the skull in such a way that its external coverings cannot keep pace with it. He wrote: "Studies of adult growth show that in brainworkers where the circulation of the organ is enhanced, the

brain continues to grow through the fifth decade or longer and many intellectuals are bald, but idiots and morons are seldom so. We believe that the differential adult growth leads to tension, to thinning, and to ischemia of the scalp with resulting loss of hair. Primitive man was not afflicted thus nor do women with their smaller brains suffer such a fate."

Szasz and Robertson commended Schein and Young for pointing out the role played by tension in the etiology of alopecia and for accounting for the specific distribution of ordinary alopecia by local anatomic factors. They criticized Young for referring to ordinary male pattern alopecia as senile alopecia and for stating that women do not become bald because of their smaller heads and brains.

Observations on the facial expressions of bald persons stimulated Szasz and Robertson to advance some hypotheses concerning the role of tension of the scalp muscles in the etiology of ordinary human alopecia. Their theory is based on some early observations of Darwin's on the defensive significance of retraction of the ears in animals that fight with their teeth. They showed that there is an intimate association, both from the point of view of embryologic development and innervation, between the muscles of the face, ears, and scalp, and deduced that ordinary alopecia is the end result of chronic activity of the scalp muscles (via branches of the facial nerves) which leads to shearing stresses in the dermis of the scalp and consequent ischemia. The specific pattern of ordinary baldness is accounted for by the anatomy and mechanics of the structures involved. These observers state that in man's phylogenetic past this pattern of muscular contraction served a defensive purpose. Now, though biologically nonadaptive, it persists in some persons as an expression of defensive attitudes. According to them the role of the male sex hormone in baldness is related to its effect on the distribution of body fat. Women and children have a thicker subcutaneous fat padding in the scalp, which resists the blood vessel pinching effect of shearing stresses. Androgens in reducing the thickness of this cushion, are thus one of the prerequisites for the development of ordinary alopecia. They also state that genetic factors probably act by predisposing to certain skull shapes which favor the development of great tension of the scalp or possibly by determining affinities for more archaic kinds of muscular expression. Electromyographic studies were undertaken by Szasz and Robertson in an attempt to provide evidence for this thesis. The results based on experiments of an essentially brief nature, did not reveal significant differences between the scalp muscle activity of bald and of non bald persons.

It follows from this view that any interruption of the nervous stimuli to the muscles involved if performed before irreversible local changes have occurred, would serve to arrest the process and possibly lead to regrowth of hair in areas only partially damaged. Practically injection of procaine or alcohol into the branches of the facial nerves innervating the occipitalis muscles might achieve the desired result temporarily. bilateral section would provide a more permanent and complete type of severance. Only those branches of the facial nerves which innervate the occipitalis muscles need be sectioned; this should be a relatively simple and harmless procedure according to Szasz and Robertson. I am very skeptical as to the value of such a procedure.

Light recently published an article on histologic studies of human scalps which were obtained during autopsy shortly after death due to unnatural causes including many types of accidents and homicide. Light found that with increasing age there was (1) a tendency toward a decrease in the number of hairs in the crown area (of males) and (2) an increase of connective tissue infiltration into the fat layer of the skin in the same areas. He stated that "there is a definite trend to lack of hair when the connective tissue infiltrates the fat layer and decreases its volume in the region where the hair root grows." In other words, male pattern alopecia is associated with an increased density of connective tissue in the region of the hair root.

Light concluded this article with the speculation "If it is assumed that in restricted areas certain unusual physiological processes are at work for a time before actual baldness is evident, the measurement of these processes would be very helpful in predicting baldness and might lead to preventive measures." The same author subsequently performed a series of experiments based on the measurement of the force required to extract a single hair. The measurements obtained so far were of no value inasmuch as their correlation with baldness was concerned, although further refinements in technique and the elimination of extraneous factors may render future studies meritorious.

According to Hamilton, the primary factor necessary for the development of common baldness or of lesser amounts of hair loss is an inherited predisposition. He states that members of certain races inherit tendencies either to retain hair or to exhibit certain patterns of alopecia. The influence of inheritance is shown also by the fact that the eunuch whose normal male relatives are not bald does not acquire baldness himself upon treatment with androgens in amounts that are more than adequate to induce baldness in other eunuchs whose normal male relatives tend to be bald.

Another factor of importance in the induction of ordinary baldness is endocrine stimulation, particularly that provided by testes and ovaries (see endocrine physiology, page 98). Patterned hair loss failed to appear in males who were castrated prior to puberty and in those who for other reasons, did not mature sexually. Androgens appear to be the agents usually implicated in common baldness, especially in extensive baldness, which appears normally only in men. Alopecia does not develop in men who fail to mature sexually but can be induced by androgenic treatment of eunuchs whose pedigrees show them to be susceptible to baldness. Whether the type of hair loss which develops in a few women after sexual maturation is due to androgenic components of ovarian, adrenocortical and other secretions or their metabolites, is not known at present, although the possibility is a good one.

A third factor is aging. In normal men advancing age is accompanied by increase in the incidence and extent of baldness. In eunuchs who were castrated prepuberally and given androgenic treatment at a later date, those who are in the second decade of life when treatment is begun tend to lose hair slowly over a period of years as in most normal young men of their age. In contrast, the eunuch who reaches the sixth decade of life before receiving androgenic treatment loses his hair

within a few months following initiation of treatment. Evidently the susceptibility to alopecia increases with age but is not expressed in the absence of inciting agents (i.e. androgens).

These genetic, endocrine and aging factors are interdependent. No matter how strong the inherited predisposition to baldness, alopecia will not result if inciting agents such as androgens are missing. This is illustrated by the luxuriant scalp hair in old eunuchs who were castrated prior to sexual maturation. Neither are the androgenic inciting agents able to induce baldness in individuals not genetically disposed to baldness. The augmenting action of aging has been referred to above.

Hamilton's studies showed that although androgens induce common baldness, gradations in the degree of baldness among normal men are not accompanied by parallel gradations in the quantity of urinary ketosteroids or in the amount of growth of a secondary sex character, axillary hair which reflects the degree of stimulation afforded by these steroids. Instead there is the paradox that the androgen-dependent condition of common baldness increases in frequency and extent with age whereas the quantity of these stimulating agents decreases with age. The explanation of this paradox is of some importance since baldness is a condition which may be paralleled to some extent by many of several hundred pathologic conditions that select males preferentially and occur with increasing frequency with advance in age. Pathologic conditions of this type contribute materially to the lesser viability of males than of females which in the United States at the present time amounts to an average of five years' shorter life span in males than in females. Hamilton draws the conclusion that although the quality of maleness is the basis of this sex difference in viability and in susceptibility to a host of pathologic conditions, the role of testicular secretions is but a setting of the stage, the action on which is controlled by genetic, aging, environmental and other factors.

It has long been contended that seborrhea, the "seborrheic state" and seborrheic dermatitis are causative agents and even the causative factors in the production of ordinary baldness or male pattern alopecia. In the light of modern research, this theory is no longer tenable. Ordinary baldness or patterned alopecia is due in the vast majority of cases to an inherited predisposition, hormonal factors, the normal aging process and in minor degree to other physiologic mechanisms such as increased tension of scalp musculature. The association of the various seborrheic states with patterned alopecia is more than coincidental, however, because the same hormonal incitant to baldness, namely androgen, also plays a part in the development and aggravation of this group of diseases. In other words, both male pattern alopecia and seborrheic disorders may be induced and aggravated by a preponderance of male sex hormone. Once this association has occurred, it is my belief that the type and amount of hair loss may be exaggerated and increased by the seborrheic components of the process. Further evidence in support of my belief has been advanced by the recent work of Flesch (see *Sebaceous Glands*, page 54). He showed that squalene, oleic and linoleic acids, normal components of sebum, cause reversible baldness when applied to the skin of laboratory animals with profound changes in epidermal growth and keratinization. In a recent personal communication, Flesch reiterated the known facts that androgens are essential for the development of common male baldness; on the other hand, male sex hormones are also the

most powerful known stimulants of sebaceous secretion. The latter secretions, *per se*, may actually be responsible for baldness although Flesch has been unable to depilate human beings with any of these unsaturated compounds. As yet, we can only say that the association between male pattern alopecia and the seborrheic diatheses is of considerable interest. That this association is neither fundamental nor necessary is evidenced by the many individuals with seborrheic changes who show no signs of baldness. Then too, the seborrheic soil is frequently observed in women, yet baldness is comparatively uncommon. Still another fact is the frequent appearance of seborrheic dermatitis over the occiput and in the eyebrows without loss of hair in these areas. This does not mean that seborrheic dermatitis may not produce alopecia. It may not only produce alopecia, but in some instances of severe seborrheic dermatitis, alopecia of an extensive and cicatricial nature may be observed. Nevertheless, it should be reiterated that the seborrheic disorders cannot be assumed to play a role of fundamental importance in the production of ordinary male baldness.

I also believe that many local diseases of the scalp and many systemic and glandular dysfunctions can both initiate and aggravate the processes primarily related to male pattern alopecia. In these instances, the type and degree of baldness may be more severe and extensive than would ordinarily have been the case.

Therapy

At the present stage of our knowledge, there is no effective remedy for ordinary baldness either prophylactically or therapeutically. The millstones of inherited, genetic patterns, hormonal variation, and aging grind on inexorably to a hairless scalp. If we accept scalp tension as a factor, then there is some logic to the prevalent usage of therapeutic massage. Yet, patterned alopecia affects scalps that are loosely adherent to the underlying tissue as well as those that are 'tightly' bound down. And there have even been reports of partial alopecia occurring as a complication of excessive massage, with regrowth following cessation of the latter. Nevertheless, gentle massage of the scalp rarely does any harm and proponents of the tension theory argue in its favor.

The use of hormonal agents in the treatment of ordinary baldness is still in the experimental stage. Although numerous reports of regrowth of hair following the use of some endocrine secretion are frequently circulated, there is no hormone as yet which can either govern or prevent patterned baldness. In select instances of glandular aberration appropriate hormonal therapy is effective, but these measures must be restricted to specific and well studied cases (see Chapter I, section on endocrine physiology). There are no indications for the indiscriminate use of thyroid, pituitary, estrogen or androgen extracts, either locally or parenterally, for the express purpose of stimulating hair growth in male pattern alopecia.

Local applications of various drugs, rubefacients, chemical irritants, physical modalities, and the like have never proved of value in the prevention or treatment of ordinary baldness. In cases associated with local scalp disease such as the seborrheic diatheses, it is my belief that proper local measures directed toward control and cure of these diseases will lessen the eventual degree and extent of alopecia. These measures are discussed under the headings of the individual diseases.

4 CICATRICIAL ALOPECIA DUE TO PHYSICAL AGENTS

A scarified bald patch due to trauma usually has a sharply defined linear or angular outline. This characteristic would aid diagnosis even if the physician were unaware of the patient's history which explains the origin of the scalp tissue cicatrization resulting in permanent alopecia.



Fig. 87—Traumatic alopecia following local injury (Courtesy Dr. F. Combes)

MECHANICAL

Blows on the head which cause open wounds are responsible for many of these denuded areas. They may be suffered through the patient's falling against a hard unyielding surface, or may be the result of an accidental blow delivered to the scalp from an unexpected source. Children suffer such wounds when they are playfully or maliciously hit over the head with croquet mallets, are cut by implements during the course of mock war, or slip because of unsure footing and hit their heads on rocks, curbstones or even the sharp edges of furniture. Adults may be injured by flying glass and the twisted steel of an automobile accident by insufficiently guarded or carelessly manipulated machinery in a factory (complete traumatic avulsion) by wounds received during war or any other of the thousand and one possible injuries of the machine age in which we live. But no matter what the cause, the heal

ing of these wounds may involve the death of the hair follicles and the formation of scar tissue with ultimate alopecia due to mechanical trauma. In diagnosing a cicatricial lesion of the scalp, the failure of the patient in recalling an early traumatic episode must always merit consideration by the examiner.

Traumatic Marginal Alopecia (*Alopecia Luminaris Frontalis*)

Clinical Features—This disease is usually located in symmetrical triangular areas at the periphery of the hairy scalp in front of the ears. The loss of hair may be complete or partial, and in cases of long duration the follicles become atrophied and cicatricial alopecia of a permanent nature results. When first seen by the physician, the affected plaques may retain signs of erythema and folliculitis, or may already display atrophy of the follicles.



Fig. 88.—Avulsion of the scalp hair by an electric drill. Note irregular limits of patch of alopecia. (Courtesy McCarthy: *Diseases of the Hair*.)

The disease often has its onset around the time of puberty as two pruritic, erythematous patches above the ears. This subacute process undergoes recurrent episodes of an inflammatory and infectious nature characterized by erythema, scaling, pustulation, and crust formation. As a result, the hair follicles are destroyed and then superseded by triangular bald patches with minute areas of cicatricial alopecia. The triangular areas are always similarly patterned. The lower angle lies approximately opposite the tragus; the sides are formed by a line of peripheral hair, while the base is the straight margin formed by the normal long hairs pulled up tightly from the temples and secured by combs or hairpins. This strip is due to the fact



Fig 88 —Triangular area of alopecia involving the temporal region anterior to the ear and due to constant traction from the use of tight curlers (Courtesy Drs S Ayres, Jr, S Ayres, III, and J Mirovich Arch Dermat & Syph)



Fig 90 —Area of traumatic marginal alopecia above ear due to constant traction from tight braids (Courtesy Drs S Ayres, Jr, S Ayres, III and J Mirovich Arch Dermat & Syph)

that short hairs cannot be caught up by the comb or pins exerting the traction and so escape a like fate. It is possible that this same condition may spread across the forehead of the patient, as the process may extend anteriorly in two narrow, ribbonlike bands which meet at the midline.

For many years all the cases reported were confined to Negroes until Riburo's white patient seen in 1938 later followed by five other cases described by Ayres and Mirovich. An 18 year old girl was observed to lack the normal amount of hair on the sides of her scalp above the ears. This area of partial alopecia was interspersed with small follicular papules and pustules. Another girl of the same age displayed a moderate thinning of the hair in the temple region. Several broken hair shafts were still visible, some of them exclamation point in type, and scattered between them were follicular pustules. The first patient was in the habit of braiding her



Fig 91—Traumatic marginal alopecia extending as a band across the forehead (Courtesy McCarthy Diseases of the Hair)

hair tightly and then pinning the braids securely across the top of the head exerting unnatural and destructive traction on the hair. The second girl was found to have been using metal curlers around which she tightly wound strands of her hair, and the resultant trauma had been in evidence for six weeks. Another victim of the metal curler was a patient of 65 years who had well developed areas of atrophic alopecia with a triangular form on either temple still displaying in their periphery a few broken hairs and gaping follicular orifices. Above the denuded patches could be seen erythematous ridges marking the site of the metal curlers. A child of 11 with a compulsive habit of brushing the hair continuously was seen with pronounced thinning of the hair in association with an inflammation

of the temporal regions on which several crusted papules were observed, as well as broken hairs. A fifth patient who had had continuous hair fall for a year and a half, still showed erythema at the temples and hairless follicles blocked with keratotic plugs. The entire area, which had been subjected to demonstrable trauma, showed evidence of an advancing atrophic change in the scalp tissue.

Pathology—Perifollicular erythema and eventual folliculitis with resultant cicatrizing of the involved area is the pattern common to all such cases.

Etiology—The conditions described above were all traceable to continuous abnormal traction on the hair from too vigorous and repeated brushing, a tight, usually braided method of dressing the hair, and the winding of hair over curlers. These repetitive episodes result in a traumatic folliculitis. The high incidence of this disease among the Negro people is probably due to the methods used for the purpose of removing the kinks which they consider unsightly.

Therapy—While traction continues, the disease slowly progresses, producing scarring. If the trauma is interrupted in time, hair loss is checked but no regrowth is possible on the cicatrized regions.

COLD

Excessive cold and heat are also responsible for some of the cicatricial conditions of the scalp. Third degree freezing following overexposure to below zero temperature displays certain well known clinical features. The skin becomes bluish black in color sometimes with bullae on its surface. The resultant necrosis is due to vascular changes. After a brief interval the dead tissue sloughs off, showing either moist or dry gangrene. Then the usual slow granulation process fills in the affected areas with scar tissue and no regrowth of hair on the denuded patches may be expected. This sequence of events may be observed, in lesser degree following therapeutic applications of solid carbon dioxide to the scalp.

HEAT

Third degree burns caused by contact with dry heat and third degree scalds from steam are equally responsible for cicatricial alopecia. They are far more numerous than those due to freezing and are usually more severe and more extensive in area. The eschar created by such a burn is worse than the destruction of the blood vessels but not quite as serious as carbonization.

Accidental contact with steam, hot objects and boiling liquids resulting in third degree burns, at first leaves the scalp resembling a piece of pale leather with no obvious inflammation nor loss of epidermis or hair. After a period of from three to five days, however, a plaque becomes evident which is bordered by an erythematous margin several millimeters in width. Within ten days to two weeks the leathery eschar becomes loosened and is sloughed off. Then the process of epithelization begins progressing from the border inward to the center until the atrophic, hairless scar is complete. Such deep scarring tissue has a tendency to become keloidal. Fourth degree burns involving various degrees of carbonization not only cause permanent alopecia but are also prone to develop carcinoma of the affected regions.

Carcinomas involving burned scalp areas are almost invariably of the squamous cell type. They are slow growing, highly differentiated neoplasms. These lesions are radioresistant, as are most neoplasms involving scar tissue, because the insufficient blood supply interferes with the normal process of repair of the tissue reaction following radiotherapy. In addition, constant infection of ulcerated, burned areas interferes with successful irradiation, and both radionecrosis and sloughing occur quickly following relatively small doses. The treatment of choice in carcinoma of the scalp involving scar tissue is wide, radical excision followed by skin grafting.

ULTRAVIOLET LIGHT

Ultraviolet light used for therapeutic purposes may be a causative factor of a superficial burn. The longest wave length capable of burning is at 3,150 Å, but maximum ill effects are produced at a wave length of 2,967 Å.

Ultraviolet therapy is often used to produce moderate erythema and superficial exfoliation of the scalp. When properly utilized under medical supervision, it has certain specific effects and may be of definite value. Excessive exposure to natural or artificial ultraviolet radiations, especially in scalps with little hair protection, may result in burns of a moderate to severe degree. These burns, resulting from excessive or unsafe wave lengths, heal slowly and may eventuate in the formation of scar tissue with minimal or no regrowth of hair from the atrophied follicles.

ROENTGEN AND RADIUM RAYS

Permanent alopecia has also resulted from carelessness in the use of roentgen or radium rays. Accidents have occurred with improperly calibrated machines or because operators have neglected to watch the exposure time or have omitted filters when they were necessary.

Alopecia has also occurred when too high a dosage has been employed for the initial treatment, when too many small doses have been given over a long period of time, and when there has been an overlapping of the radiated fields even when the correct dosage has been used.

Some individuals (blondes with fair complexions) have a radiosensitivity beyond that of the normal person. It is because of these patients that it is particularly important to begin a course of treatment with very low dosage, increasing it slowly until one can be sure that the patient's reaction equals normal expectancy. The scalp is more susceptible to x ray damage than other parts of the body since it is relatively free of the protection offered by subcutaneous fat (for discussion of x ray epilation, see page 324).

In general x ray therapy of the scalp is confined to the performance of an x ray epilation for tinea capitis. However, in some instances of localized disease of the scalp x ray therapy is of value but should be limited to doses of low voltage and minimum r/air value (35-75 r weekly for a maximum of 4 doses). In recent years, dermatologists have found ultrasoft (Grenz) rays particularly adaptable for the therapy of resistant localized scalp disease because of their low degree of pene-

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within forty eight hours following the x ray exposure as a deep dusky erythema. The affected site rapidly shows a brawny edema often with scleroderma like hardness within ten to fourteen days. This area of solid edema usually sloughs out to form a deep and extremely painful ulcer, although occasionally it may remain as a hard crusted patch of dry gangrene. These lesions are resistant to therapy and heal very slowly, if at all over a period of many months or years. Long standing ulcers may go on to the formation of squamous cell carcinoma exhibiting a spindle cell metaplasia of a high degree of malignancy. These carcinomas metastasize relatively early to regional lymph nodes. The proper treatment of such lesions is either wide surgical excision with immediate application of whole thickness skin grafts or electrocoagulation followed by endotherm excision and secondary skin grafting.

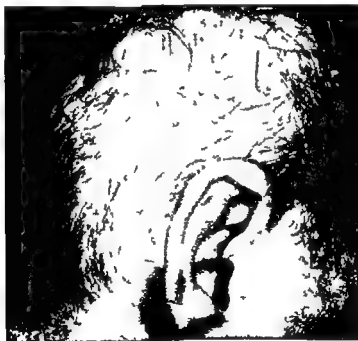


Fig 97—Cicatricial alopecia as a manifestation of radiodermatitis following x ray therapy for alopecia areata. X ray therapy should not be used as a method for inducing or stimulating hair growth. (Clay Adams Co.)

The scars following a third degree ulceration always show complete alopecia. In some instances the cutaneous healing may be sufficient to restore the bald patch to the color and consistency of normal skin but in the usual instance the involved site shows the typical sequelae of chronic radiodermatitis. On the scalp chronic radiodermatitis is manifest by telangiectasia (dilated cutaneous vessels), atrophy (thin shiny wrinkled dry occasionally sclerodermatous), pigmentation (both hyper and hypo), loss of sweat and sebaceous glands, keratosis, late ulceration and skin cancer. Any or all of these reactions may occur following a first degree reaction but are most often encountered following a second or third degree reaction to

x-rays or radium Alopecia of a complete and permanent nature usually follows second and third degree reactions. However, it is important to recognize the fact that permanent alopecia may occur as a sequel to even a mild first degree reaction. It is even possible to produce permanent alopecia following repeated exposures of the scalp to small doses (75 r) of x-ray. Loss of hair following exposure to the x rays or radium is usually permanent if no regrowth has occurred after the passage of a six month period following the exposure. If regrowth does occur, it usually appears within one to six months, depending upon the type of exposure and amount of x ray administered. I have seen partial regrowth of hair following second degree x ray reactions, but this is uncommon.

At this point, it is advisable to discuss the use of x rays for the purpose of stimulating hair growth. The suggestions for such usage may be found in many textbooks and in the general literature on the subject of hair. It is my opinion, as well as that of the majority of students and investigators in the field of x ray and radium therapy, that there is absolutely no indication for such a procedure. There has never been any corroborative evidence to the effect that the radiotherapeutic modalities are in any way capable of stimulating hair growth. The statement is also made that, subsequent to x ray epilation the returning scalp hair was of better quality, texture, and luxuriance than prior to the x ray exposure. There has never been any conclusive proof of such observations, and categorically, it may be stated that if anything, the hair growth is less luxuriant and of poorer quality.

ATOMIC BOMBS

Liebow described the effects on the scalps of Japanese people exposed to atomic bombs. Clinically, epilation usually appeared rather suddenly, approximately two weeks after the irradiation, chiefly among persons who had been within 1,500 yards of the hypocenter, the point on the ground above which the bomb exploded. It affected men, women, and children alike. With very few exceptions, the distribution was that of ordinary male pattern alopecia: the temporal and occipital regions being relatively spared. Even when epilation was almost complete, a few hairs tended to remain in a very sparse arrangement. In a very small number of persons whom distance or shielding brought to the outer limit of the range of the ionizing radiations, epilation involved only that side presented toward the bomb. The hair of the brows, lids, beard, and body was only rarely lost, despite almost total epilation of the scalp. Almost all patients whose death could be attributed directly to radiation effects, and who lived for more than two weeks, manifested epilation. The mean day of onset of epilation was the twenty fifth. Some individuals complained of dryness of the epilated skin—a symptom difficult to evaluate, since anhidrosis was not complete. Among survivors the hair usually began to reappear in some measure between the sixth and eighth weeks. Recovery was progressive, with ultimate restitution of the hair in its former abundance, color, and texture by the fourth or fifth month. The persistence of alopecia beyond this period of time was considered an important sign indicative of a poor prognosis as far as the life of the individual was concerned.

Liebow observed that the histologic changes were similar to those observed in the normal cycle of loss and replacement of hair, except that the glassy and connective tissue sheaths were possibly thicker and the pigment more irregularly distributed in the temporarily atrophic follicles. The process of atrophy in the irradiated subjects was condensed in time and imposed upon a vast number of follicles all at once, with consequent epilation. The baldness was transient, since the survivors were comprised only of those who had received, upon the body as a whole, less than a permanently epilating dose of ionizing radiations.

5 MEDICOLEGAL ASPECTS OF HAIR MORPHOLOGY*

Specimens of hair are frequently of value from the standpoint of medicolegal examination. The careful examination of hairs found at the site of a recent murder or many other forms of crime or offense may be the determining clue in detecting the guilty party. Glaister, in his excellent book on medical jurisprudence and toxicology, discusses the problem with great care and thoroughness. In the course of his studies, he has developed several techniques and examinations which enable the examiner to distinguish the different types of hair observed in the various species, the recent presence or absence of dyes, and innumerable additional factors of importance. He states: "With experience, it is not difficult to differentiate between the hairs of man and those of the lower animals. If a hair root has been forcibly extracted the bulb will be irregular in form, due to rupture of the sheath, and will show an undulating surface, together with excrescences of different shapes and size. A naturally shed bulb has a rounded extremity, a smooth surface, and most probably will show signs of atrophic or fatty degeneration, especially in elderly persons."

In view of the number of criminal cases in which the finding of hairs on various objects and persons has presented itself, and the value of these in relation to the identity of accused persons, Glaister made a close investigation of the hairs of the entire mammalian class of animals.

"The investigation was approached from three aspects, namely: the study of hairs belonging to the mammalian group, excluding human hairs, the examination of human hairs from different parts of the body, and the examination of cross sections of all the hairs in the preceding groups. The object of research in the above scheme was to determine whether the hairs of animals belonging to the same zoological order, sub order, or family showed characteristics so similar that they could be depended upon for the identification of the animal order, sub order, or family to which the hair belonged. In the case of human hairs, to find whether there were any specific differences in their appearance with regard to sex, age of the subject, site on the body from which they were taken and pigmentation. The examination of transverse sections supplemented the information previously obtained from examination in the longitudinal plane.

A collection of hairs was made from many sources, and some 1700 photomicrographs of specimens were taken and classified by Glaister. The principal conclu-

*The greater part of this material has been obtained from *Medical Jurisprudence and Toxicology* by Professor John Glaister, published by E. & S. Livingstone, Edinburgh.

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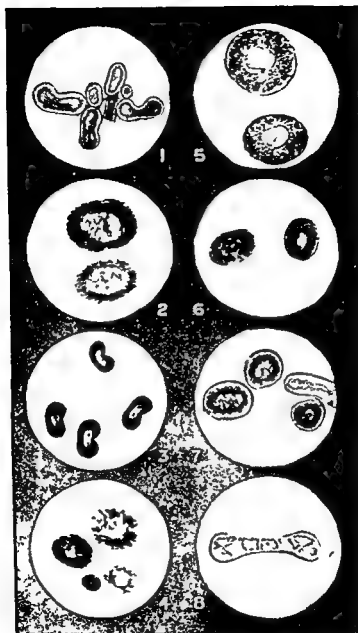


Fig 94—Cross section of hairs (1) Squirrel (2) cat (3) goat (4) dog (5) horse (6) cow (7) rat (8) rabbit. (Courtesy Professor John Glaister Medical Jurisprudence and Toxicology E. & S. Livingstone, Edinburgh)

sions drawn by him from this investigation were 'First, the appearance of the majority of animal hairs is such as to permit the identification of the order, sub order, or family of the animal to which they belong, second, the characters of the hairs of animals belonging to all the mammalian orders excepting a few belonging to the Primates or monkey families, are such that their naked eye appearances are likely to reveal that they do not belong to the human race, and that in the odd instances mentioned a detailed examination of the structure will show that the pigment in the cortical layer is coarser, and the medulla broader and less regular than in human hair third, that where doubt as to differentiation between animal and human hair should arise from examination in longitudinal plane, the appearance of the transverse or cross section is likely to settle the question by reason of the fact that the core or medulla of human hair is invariably smaller than that of any of the other Primates, fourth that as a general rule, the breadth of the medullary layer is less in the hairs of the higher than those of the lower grade mammals

From the medicolegal point of view, the examination of hairs should include the following particulars (1) color to naked eye and on microscopic examination, (2) length ascertained by actual measurement (3) texture (4) approximate breadth by micrometer (5) hair tip or hair end characters, whether intact, cut, or torn (6) condition of bulb if present, whether forcibly pulled out, degenerated, or cut across by a sharp instrument or crushed by a blunt instrument (7) character of cuticle, extent and character of cortex, presence or absence of medulla and, if present the character and breadth (8) whether hairs are dyed or undyed, (9) contour of transverse sections in respect of points set down in No 7

The detection of hair dyes is important in some cases, and its presence is indicated usually by a lack of uniformity in the color of the hair, which is often lacking in lustre and brittle in character. The natural color of the more recently grown hair at the roots strikes a contrast with the shade of the remainder of the hair on the scalp and this is well demonstrated by the use of filtered ultra violet light, when dyed hair appears lustreless. The use of infra red photomicrography, especially when the hair has been dyed or bleached is an important adjunct in the examination of hair. Comparison of the color of the hair on the head with that of the hair on other parts of the body will frequently indicate the presence or absence of dye in the former. The scalp may occasionally show staining due to the dye which has been employed. On applying appropriate chemical tests the nature of many of the dyes commonly used can be detected. A portion of the hair may be steeped in diluted nitric acid and this will give a solution which can be tested by qualitative chemical methods. On examining dyed hairs microscopically the intimate structure appears hazy and shows a uniformity in general shade which is not seen in hairs of natural color. This haziness is more marked when the darker dyes have been employed but when bleaching substances have been used such as hydrogen peroxide, the substance of the hair shows a typical bleached appearance which differs from the character of natural white hairs. See sections on Anthropology (Chapter I) and Hair Dyes (Chapter II)

Smith and Glaister also described a method for the preparation of hair specimens for examination in a longitudinal plane. They observed that prior to mount

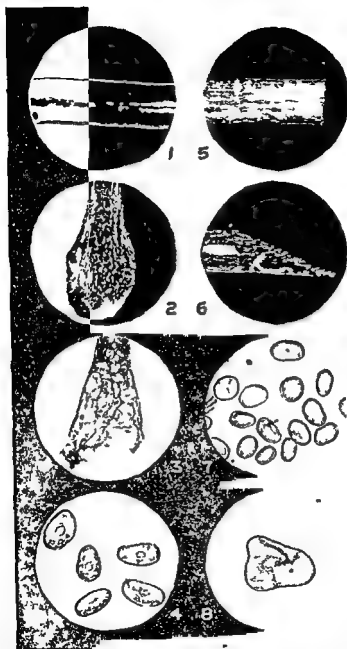


Fig 95—Certain hair features (1) Gray hair (?) healthy bulb (2) degenerate root (3) pubic hair (4) hair cut by sharp blade (5) hair severed by blunt blade (6) scalp hair (7) mustache hair (8) mustache hair (Courtesy Professor John Glaister: Medical Jurisprudence and Toxicology E & S Livingstone Edinburgh)

ing the specimens it is necessary to use some cleansing reagent to remove adherent debris which may mask structural detail. One of the most efficient consists of equal parts of ether and absolute alcohol. The specimens should be placed in a small test tube with this solution and gently shaken. The hairs are then removed from the tube and dried between sheets of filter paper. Before mounting they should be steeped in a good quality benzol or oil of turpentine which acts as a clearing agent. After drying the specimens are mounted preferably in Euparal or in Canada balsam long cover slips applied and the slides allowed to stand for twenty four to forty eight hours if time will permit for clearing purposes.

The following are the stages recommended for the preparation of cross sections of hairs

- 1 Cleanse hairs in equal parts of ether and absolute alcohol
- 2 Place in a solution of 2 per cent alcohol to which has been added an equal quantity of 5 per cent liquor ammonii fortis or ammonium hydrate. Steep for five minutes
- 3 Soap in 10 per cent potassium hydrate for one or two minutes. Maintain temperature at 50° C
- 4 Wash specimen in a solution composed of equal parts of 5 per cent sulphuric acid and absolute alcohol for some minutes
- 5 Wash in xylol or benzol for a few minutes
- 6 Dry hairs between sheets of filter paper
- 7 Steep hairs in liquid paraffin wax for fifteen minutes
- 8 Block specimens in paraffin
- 9 Cut sections about 8 to 10 μ in thickness
- 10 Float sections on water at temperature of 40° C
- 11 Place on albuminised slides and put in incubator at temperature of 37° C for twenty four hours
- 12 Dissolve paraffin wax with xylol dry slides and add Canada balsam or preferably Euparal

The objects of stages 1 to 5 are to cleanse soften and dehydrate the hairs step 7 to ensure adherence of paraffin during blocking stage step 10 to uncurl the sections and step 11 to ensure adherence of the sections to the slides

A variety of paraffin waxes of different melting points should be available in the ovens since success lies in the selection of a wax of suitable melting point having regard to the thickness and consistency of the hairs to be sectioned. For general purposes a wax of melting point at 52° C should prove satisfactory. During the various stages the hairs are immersed in the form of a bundle. The bundle is made by binding the strands together at intervals with fine silk thread. When only a single hair is available the various stages may be carried out in a watch glass and the specimen embedded by means of fine forceps.

Kneberg introduced a method for embedding hairs for sectioning purposes. According to Glaister. The hairs are mounted on a piece of three ply drawing card board 4 1/2 by 3 cm. Slanting cuts are made in the ends of the cards and an opening 2 by 1 cm made in the centre of the card. The hairs are slipped through each end and are then held taut by small rubber tipped clamps until the glue which fastens

them, is dry. Fish gelatin dissolved in warm water is recommended, since it is insoluble in the alcohols. When the specimens have been put through the various stages, and are finally embedded, the block is cut down leaving only the part containing the hairs. It is also advised that, during the cutting of sections, the microtome blade should be kept moist with 75 per cent alcohol to obviate curling of the sections" (See page 68)

In 1937, Moritz, when working in Glaister's laboratories, performed several original investigations in connection with cuticular scale impressions. Experimentation then showed that the most simple and suitable method for making such impressions was to use a plastic film of cellulose acetate in the form of fingernail polish diluted with an equal quantity of amyl acetate. A drop of this solution should be spread thinly over the surface of a microscope slide, and the cleansed hair, with some tension exerted upon its ends, should be laid over the wet film. After the film has dried, usually in about ten minutes, the hair is stripped off the slide by a quick movement. The scale impressions are then examined microscopically. This method is very useful in the instance of hairs which are deeply pigmented and when examination of the cuticular scales is desired. The color of the polish affords clear definition of the structural detail and facilitates photography.

In 1945, Chief Inspector George Maclean and Detective-Inspector Charles McNeill, both of the Identification Bureau, City of Glasgow Police, experimented with all the known formulas for the media employed in the preparation of cuticular casts, and finally originated the following formula, as described by Glaister

Ethyl lactate	-----	5 millilitres
Amyl acetate	-----	39 millilitres
'Cellodin'	-----	6 grammes

'The excellence of the results obtained, in addition to the fact that the solution is equally suitable for hairs of all textures, justifies the recommendation that this plastic medium should invariably be used in making hair casts'

Davidson and Taylor also described a method for staining the cuticular cells of hair. The root and tip of the hair are removed, the hair is placed in equal parts of absolute alcohol and ether for fifteen minutes, and then washed thoroughly in distilled water. Next, the specimen is placed in distilled water in a vacuum embedding oven at room temperature, and a pressure of 18 to 39 mm mercury is maintained for two hours. This process ensures the withdrawal of air from the medullary part of the hair. The specimen is now bleached in a solution composed of 50 volumes hydrogen peroxide, diluted 1 in 3 with distilled water, 50 ml, 5 per cent aqueous ferric chloride, one drop, and excess of liquid ammonia which is added immediately before use. The stage of bleaching occupies a period of from fifteen minutes to twelve hours depending upon the degree of pigmentation of the hair. The specimen is washed thoroughly in distilled water and stained for at least fifteen minutes in 1 in 100 carbolfuchsin (Ziehl-Neelsen) in distilled water in a vacuum embedding oven at room temperature and at a pressure of 18 to 30 mm mercury. The hair is then washed in distilled water, decolorized in absolute alcohol for five to ten minutes, cleaned in benzine and mounted in Canada balsam. The writers assert that this method permits the study of the cuticular scales and the longitudinal

section of the hair in detail, and that, with the use of suitable plates and color filter, photographs showing maximum detail and contrast can be obtained (See Chapter I, section on Anthropology, for additional reference)

Sometimes it is desirable to prepare stained preparations of hair or epithelial scales for mounting. The following method (Berberian) has been recommended

Technic—First, cut scales or hair into small pieces (1 to 2 mm) and fix on slide by covering with 50 per cent glacial acetic acid in water, which is allowed to dry in an incubator

1 Defat specimen by flooding two or three times with ether, which is allowed to remain 20 to 30 seconds each time. Then flood slide for 30 to 60 seconds with absolute alcohol, followed consecutively by 95, 70, and 50 per cent alcohol

■ Stain for three to five minutes with Martinotti's toluidine blue

Formula:

Toluidine blue	-----	10 Gm
Lithium carbonate	-----	0.5 Gm
Distilled water	-----	75 cc

After dye has dissolved completely, add 20 cc of glycerin and 5 cc of 95 per cent alcohol

3 Wash slide in water and differentiate in 0.5 per cent acetic acid

4 Dehydrate in acetone, clear in xylene, and mount in balsam

Additional techniques for staining and mounting hairs are described in the section on Anthropology, Chapter I

6. PSEUDOPELADIE

Clinical Features

The patient, a man between the ages of 20 and 45 years, or his barber usually makes an accidental discovery of this disease, because it is normally without symptoms of any kind although a few atypical cases have reported slight itching before hair fall. The alopecia develops slowly and the areas affected are at first so minute that they escape detection. By the time a physician is consulted, these small bald spots located in the vertex, temporal, or parietal regions of the scalp have begun to coalesce into cicatricial, finger shaped areas about a half-inch in length. This appearance has been likened to that of footprints in the snow. These in turn, after another period of years, merge to form a palm-sized area of atrophy and alopecia with irregular margins and intervening tufts of normal hair marking some of the borders of the smaller, irregular patches. In no case does the alopecia ever involve the entire scalp.

The denuded areas present the appearance of translucent, shining tissue, suggesting the term "onion skin." They are glossy, white, ivory, or faintly erythematous in color, with a total absence of follicular openings and somewhat depressed centrally. The hairless regions have a softer, more flexible consistency than the adjacent scalp, which McCarthy believes is due, not to atrophy, but simply to absence of hairs acting as a "stiffening support" to the scalp. Several writers have noted the lowered sensitivity of the atrophied surface as when an anesthetic is injected before performing a biopsy.

There is no obvious perifollicular inflammation present in the marginal areas, but hairs may easily be epilated with their hyaline, translucent, epithelial sheath still attached, and there is then sometimes a minute vestige of inflammation at the follicular mouth, which disappears immediately upon pulling of the hair or spontaneous hair fall. The shaft is often irregular and heavily pigmented, and the root, because of its rapid fall, seldom exhibits the tapered end found in normal exchange. There are no transitional hair types at the margins of the plaques such as exclamation point, downy, or broken hairs. On the contrary, there is a sharp demarcation between the atrophied area and the normal hair bearing scalp.



Fig 96—Pseudopelade. Lesions have been likened in appearance to footprints in the snow. (Courtesy McCarthy: Diseases of the Hair.)

Differential diagnosis from the clinical aspects of the disease offers little confusion where alopecia areata is concerned since this type of baldness is impermanent and does not result in surface scar tissue. With atrophy absent, hair follicles are present and there is no fundamental scalp change in alopecia areata. There are also fewer but larger plaques with a notable regularity of border, the presence of exclamation point hairs and no mucoid sheath around those which have been epilated.

Another type of alopecia, the syphilitic, usually exhibits freedom from atrophic change and absence of root sheaths, but when serologic reactions are negative and there are no concomitant symptoms, this type is not easy to diagnose. Early recognition is obviously essential since at the time this alopecia may be the sole symptom of syphilis (see that disease in Chapter V).

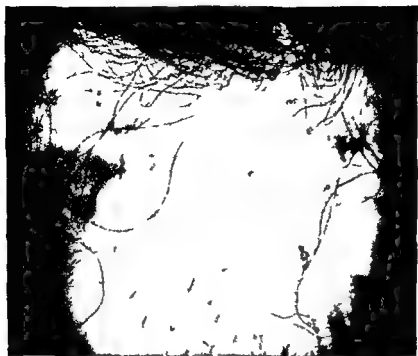


Fig. 97 Advanced stage of pseudopelade. The crusted lesions are merely artefacts due to self-inflicted excoriation. (Courtesy Dr. F. Combes.)



Fig. 98—Typical epilated hair of pseudopelade with hyaline translucent epithelial sheath.

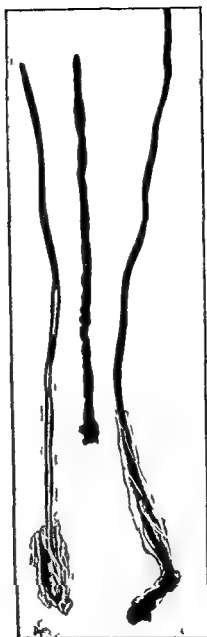


Fig. 99.—Twisted atrophic and deeply pigmented hairs of pseudopelade. Such hairs are found on the edges of the plaques. They are easily lifted out of the follicle and retain the follicle lining which forms a large translucent sheath around the lower third of each hair. (Courtesy McCarthy. Diseases of the Hair.)

Lupus erythematosus may be differentiated from pseudopelade because it never presents so many small patches in one area and, instead of an absence of follicular openings, exhibits enlarged ones stopped up with keratotic plugs. Obvious inflammation, telangiectasia, and infiltration assist diagnosis as well as the location of lesions elsewhere on the skin.

Folliculitis decalvans in its end stages is most difficult to differentiate from pseudopelade. It is necessary to obtain a history which will reveal a marginal perifollicular inflammation and frank pustulation at one stage in the disease's development. Also in folliculitis decalvans, one small plaque enlarges its own limits rather than having the final bald area result from the coalescence of smaller patches. Some authors prefer to consider pseudopelade a lesser inflammatory species of the same disease, particularly since the periodic activity and regression are common to both. The possibility exists that pseudopelade is merely an end stage of any one of several diseases capable of terminating as a cicatricial alopecia.

The healed case of favus resembles pseudopelade because of the irregular borders of the plaques, the lack of evidence of an inflammatory process, and the obvious cicatricial condition of the scalp. The preceding history is important. As are past cultural studies, microscopic examinations of the hair shafts, and the presence of a scutulum.

Pathology

Until Laymon and Murphy's excellent and exhaustive project, we had the advantages of extended studies by Brocq, Robinson, Sabouraud, Darier, Ducrey, Civatte, Van der Meulen and others, agreeing with each other in certain fundamental findings but differing in detail. Laymon and Murphy have deepened our understanding of the subject under discussion by performing biopsies of sections of the scalp removed from the vertex and occipital regions of normal specimens, of those who developed ordinary male baldness, and of those whose scalp had been denuded in plaques following tinea capitis. The cicatricial alopecia sections were then meticulously compared to the above and interesting findings were noted particularly in the cases of pseudopelade, folliculitis decalvans, and lupus erythematosus which with scleroderma, atrophic lichen planus, lupoid syphilis, and atrophic alopecia following acute seborrheic eczema are difficult to diagnose. These findings which make it possible to speak with some authority about the differential diagnosis of these diseases will be discussed under each separate heading. This may simplify the diagnostic procedure when applied to pseudopelade and other cicatricial alopecias which present a confusing similarity of appearance after their courses have been run and only the atrophic plaque remains.

Brocq, Lenglet, and Ayrignac discovered that even before hair fall in pseudopelade, there was an infiltration of the fibrous follicular covering and external epithelial sheath by a small number of lymphocytes which later multiplied to form a coating for the vessels of the papillae. Darier in his investigations found mast cells and chromatophores in association with the lymphocytes. Laymon and Murphy found small collections of these lymphocytes in their general survey of sections from advanced lesions distributed mainly about the blood vessels of the cutis and the sclerotic remnants of the follicles.

This indication of a low grade inflammatory perivascular infiltration, involving the capillaries of the cutis around and between the follicles, caused the destruction Sabouraud believed that all the tissue was involved except the erector pili muscles, which he stated were sometimes the only indication left that a follicle had existed at that point. Laymon and Murphy found that not only the muscles but the sweat glands as well persisted, although the latter showed cystic dilatation. Other writers noted that the sweat glands were distorted and compressed by the cicatrizing process surrounding them. Laymon and Murphy were unable to find in any section a complete homogenization of the connective tissue.

All writers agree that the disappearance of the follicle and its sebaceous gland is followed by the growth of young connective tissue, mentioned by Civatte, which ultimately forms a cylindrical mass at the site of the previous follicle. These columns of sclerotic tissue are perpendicular to the surface and are one of the three chief points common to all sections examined by Laymon and Murphy. Another characteristic was that in all cases they found the epidermis thinned to three layers of cells with the rete pegs flattened.

Civatte and Brocq both mentioned that dilated follicular mouths choked with keratotic plugs sometimes remained after the follicle itself and its sebaceous gland had disappeared. Laymon and Murphy found no plugging by any horny material, and it is possible that these sections reported by Civatte were not examples of true pseudopelade. These recent investigations also failed to find the hyaline blocks reported by Civatte and Meiren located in the papillary portion of the cutis. Such evidences of hyaline content would change our conception of the etiology of the disease, indicating, as it would, an endocrine or internal toxic causative factor. Barber, for instance, on this evidence believes the cause must be a blood borne toxin.

Etiology

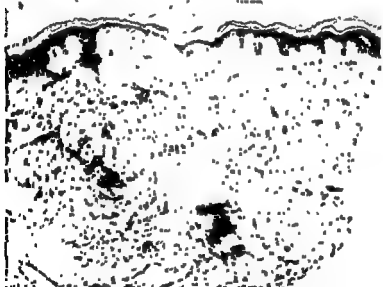
Since no organisms have been found in culture, and the presence of hyaline blocks have been recently disproved, it would appear that a low grade infection, cause unknown, initiates the scalp disease known as pseudopelade. The faint, almost imperceptible rosy tint seen briefly at the follicular mouth during hair loss lends weight to this deduction, as do the pathologic findings described above.

Therapy

Most modern writers agree that while total alopecia is never a result of pseudopelade, the atrophic plaques of baldness left by the disease are so stubbornly resistant to treatment that prognosis for regrowth of hair or arresting of its slow but seemingly inexorable course is grave.

McCarthy, who agrees that treatment is usually without effect, nevertheless reminds us that attempts should be made with local and systemic treatment until the hopelessness of each individual case has been proved beyond question. He advises us to care for general health with cod liver oil, its derivatives and substitutes, and to try hypophosphites of iron and even arsenic given intramuscularly in the form of sodium cacodylate. This latter therapy employed in courses of ten injections every spring and fall for three years, he claims, benefited four patients.

A.

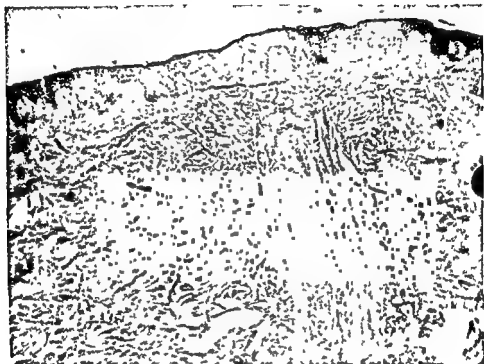


B

Fig 100—*Pseudopelade*

A, Sclerosis of follicle at extreme left. Cystic dilatation of sweat ducts ■ a result of atrophy. Loss of sebaceous glands.

B, Sclerosis of follicle with complete loss of sebaceous glands.
(Courtesy, Dr Carl Laymon and J Invest. Dermat.)



C

Fig 100, C—Only one hair follicle present in entire section. It is without hair, keratinized, and actually is a distorted remnant of an atrophic follicle. (Courtesy Dr H Curth and J Invest Dermat)

Local treatment was suggested by Sabouraud, who applied dilutions of 10 per cent sulfur in alcohol and water. McCarthy advised careful manual epilation to avoid spreading, or total x ray epilation and the following applications

	GM	OR CC
Sulfur ppt -----	2	
Resorcin -----	1	
Rosewater ointment -----	30	
or		
Sulfur ppt -----	10	
Alcohol (90 per cent) -----	20	
Distilled water -----	70	

McCarthy advises frequent shampooing during treatment to remove the sebum and allow the sulfur to penetrate as deeply as possible. He also combined oil of cade in a sulfur salve to stimulate the hair producing apparatus of the cutis as follows

	GM	OR CC
Deodorized oil of cade -----	10	
Lanolin -----	10	
Petrolatum -----	10	
Precipitated sulfur -----	1	
Oil of rose -----	qs	

He recommended that this be rubbed in for five minutes each day over the involved scalp and a half inch beyond the margin. After treatment, excess salve should be removed from the surrounding area with a soft cloth. Stelwagon suggested from 1 to 2 per cent resorcin with precipitated sulfur, and Hallopeau a rather irritating mixture composed of 5 per cent betanaphthol and 5 per cent salicylic acid. Savill, who believes that nothing is specific, recommends ultraviolet light both locally and generally.

It would seem to this writer that a careful study of the pathologic findings would eliminate any attempts at therapy if the atrophic alopecia has progressed beyond the pin dot stage. Since the disease is seldom if ever discovered in this early period and the findings are merely those of burned out tissue, time and money may be saved patients by telling them the truth and assisting them to plan a method of hairdressing which will utilize the normal growth on the scalp to conceal the cicatricial areas of baldness. Of course, further study and research must be continued in an attempt to elucidate the fundamental causative mechanisms of this disease. If such study uncovers cause and effect, then an appropriate course of action may be undertaken provided the diagnosis is established at an early stage and the disease processes are still active.

7. FOLLICULITIS DECALVANS

Clinical Features

The lack of symptoms in folliculitis decalvans usually delays consultation with the physician so that often the patient does not seek professional advice until from

five to fifteen years after its inception. Adults with coarse black hair seem to be those whose scalps are most often affected.

The original lesion, which inevitably escapes detection, is a pinhead sized, inflammatory papule or pustule surrounding the follicular orifice, and pierced by the hair shaft. One follicle after another is involved by peripheral expansion. From ten to fourteen days after the initial infiltration, crusting or desiccation occurs, with the eventual detachment of the epidermal debris. As this drops off, the hair comes with it, displaying the shiny root sheath which was once the epidermal lining of the follicle. As the hairs are shed, they leave behind a minute, reddened spot which quickly fades. There may be slight itching during the periods of maximum activity.



Fig. 101—*Folliculitis decalvans* exhibiting cicatricial alopecia. The perifollicular pustulation and erythema are typical. (Courtesy Dr. Marion Sulzberger.)

The death of these follicles and the elastic and connective tissue between them accounts for the scarring patches of alopecia which are usually evident prior to the initial examination. The center of the plaque exhibits an area of cicatricial baldness with the follicular ostia no longer in evidence. These denuded patches may range in size from narrow capsule shaped tracts to large, irregular, and ill defined oval or round spaces, but no matter what their shape or size they are bordered by reddened slightly dilated follicles capped by cones of adherent scale pierced by the affected hair. A minute pustule or groups of pustules may also be



Fig 102 —Folliculitis decalvans showing large patches of cicatricial alopecia with activity in the margins of the bald areas (Courtesy Dr W J O'Donovan *The Hair*, J & A Churchill London)



Fig 103 —Folliculitis decalvans end stage. The final burned out patches of cicatricial alopecia are similar to those observed following pseudopelade, lupus erythematosus, and favus (Courtesy Dr W J O'Donovan *The Hair*, J & A Churchill London)

apparent as perifollicular lesions. Rarely a tuft of hair will escape the spreading inflammation and remain a normal oasis, in the midst of the otherwise hairless desert that exists as an atrophic plaque. This central healing which involves the formation of fibrous tissue and permanent alopecia surrounded by active lesions in the marginal area is essential for the clinical recognition of the disease.



Fig. 104.—Folliculitis decalvans of twenty years duration. Cicatricial alopecia with the occasional appearance of localized inflammatory exacerbations is characteristic of the usual case. (Courtesy, McCarthy: Diseases of the Hair.)

Other diseases resulting in bald plaques of scar tissue may be differentiated in many ways. Alopecia areata lacks the cicatricial aspect and the marginal inflammation. Lupus erythematosus seldom appears on the scalp alone. Lesions on the face and ears, telangiectasia and less smooth scarring are usually concomitant features. Dilated follicular orifices border the denuded plaques instead of pustular folliculitis. Pseudopelade is completely free from the presence of the marginal inflammatory papules, but where folliculitis decalvans has been in process for many years and the inflammation has subsided or been controlled, the pustules are absent.



Fig 105—Folliculitis decalvans. A, Sclerosis of cutis. Obliteration of follicles and sebaceous glands. Partially emptied pustule. B, More advanced process showing epidermal atrophy, complete obliteration of sebaceous glands, and remnant of hair follicle in center (Courtesy Dr Carl Laymon and J Invest Dermat)

and differential diagnosis is impossible without either a complete history of the case or the pathologic findings. Even the latter may not clarify the diagnosis in certain long standing cases. This is equally true with scars resulting from favus, trauma, lupoid sycosis, and certain syphilids, as well as atrophic forms of lichen planus and the cicatrizing processes following severe cases of seborrheic dermatitis and other diseases.

Pathology

Quinquaud, after whom the disease was once named, found microscopically that the infiltrate was intensified around the follicle but that it was also present in the adjacent cutis and even the epidermis. Follicles and sebaceous glands disappeared as the irritative process subsided, leaving fibrous tissue in their places.

Later investigators noted that in advanced stages the stratum granulosum was either absent or limited to from one to three layers of swollen polygonal cells. As in other forms of permanent alopecia the sweat glands persisted but were without operative ducts, and while elastic tissue had completely disappeared in the upper half of the cutis, it was still evident at the level of the sweat glands.

Laymon and Murphy, in their recent pathologic study, found the central portion of the plaques identical with those of pseudopelade, but the surrounding area was characterized by abscess formation just below the basal layer which occupied most of the upper half of the cutis. In addition, there was a preponderance of polymorphonuclear leucocytes in relationship to the usual lymphocytes. All through the affected areas, the size and number of the infiltrates were larger than in average cases of pseudopelade. They did not find evidences of the intense inflammatory changes described by Van der Meiren, nor did they observe the hyalin blocks in the papillary portion of the cutis reported previously by Civatte. Special elastic tissue stains showed an absence of elastic fibers at the sites of the cicatrized follicles.

Etiology

Savill reported that *Staphylococcus aureus* was found in cultures made from the affected follicles and hair roots, repeating Quinquaud's findings but these have not been confirmed by more recent research.

Senear called attention to the many cases in which lichen spinulosis was associated with folliculitis decalvans, and wondered whether the causative factors of one might not be responsible for the existence of the other. Actually, the reference applies to an entirely different disease, namely follicular lichen planus, and the many aspects of this complex problem are discussed under that heading. (See Follicular Lichen Planus page 432.)

Therapy

Prognosis is grave and the disease is definitely a chronic one. As the lesions involute in one spot they emerge in another. While it is utterly impossible to stimulate regrowth in the cicatricial areas, many writers have found it possible to check the extension of the lesions by meticulous treatment. However, the very number of suggested methods prove that there is no specific means of therapy.

TABLE III
CICATRICAL ALOPECIAS
DIFFERENTIAL FEATURES OF PSEUDOPELADE, FOLLICULITIS DECALVANS, LUPUS ERYTHEMATOSUS, AND LUPOID SYCOSIS

NAME	PSEUDOPELADE (Occasionally and very slight)	FOLLICULITIS DECALVANS	LUPUS ERYTHEMATOSUS		LUPOID SYCOSIS
			Entire patch	Entire patch	
Erythema	No	Perifollicular	No	Severe	
Pustulation	No	Perifollicular	Yes	Yes	
Scaling	No	No	Yes	No	
Telangiectasia	No	Inflamed follicles are capped by adherent scales pierced by hair	Yes	No	
Follicular plugs	No	One or several patches (ultimately enlarge)	Usually one patch	One continuous patch often band like	
Cicatricial patches	Many small areas (ultimately less)	Yes	No	No	
Tufts of normal hair between patches	Yes	No	Yes	Yes (extends over face)	
Lesions elsewhere on body	No				

Skin of cicatricial patch during active phase of disease	Smooth, glossy (onion skin)	Smooth, white, perfoli- cular erythema of marginal hairs	Red, scaly, Telangiectatic, fol- licular plugging	Papulopustules of brown or deep red color, keloidal scars, pustular folliculitis
Infiltration	No	No	Yes	Yes
Keloidal scars	No	No	No	Yes
Hair (marginal)	Easily epilated, glassy transparent epithelial sheath attached to root,	Easily epilated, shiny root sheath still at- tached	Normal	Easily epilated, root sheath at- tached (dull, white)
Histology (After Layman)	Extreme epidermal atrophy, scler- osis of cutis with perpendicular columns of fibrous tissue replacing follicles, sebaceous glands ob- literated, erector muscles and sweat glands preserved, latter sometimes cystic, inflammatory changes much less than lupus erythematosus and composed of small collections of lymphocyte about the blood vessels and scler- otic follicles	Same as pseudopelade with addition of oc- casional abscess for mation just below the basal layer and oc- cupying most of the upper half of the cutis	Early lesions show typical hu- tologic features of lupus erythematosus, older lesions resemble pseudopelade and folliculitis decalvans with important difference that in- filtrate is heavier, and more diffuse, old lesions not diag- nostic	(After active stage is over) Atrophic epidermis, infiltrate in co- rnum composed of lymphocytes, plasma cells and rare mast cell. hair follicle and adnexae atro- phic, sweat glands present in lower corium, process essentially inflammatory, infiltrates around the follicles and leading to their gradual degeneration

Obviously all measures should be undertaken to improve the local and general resistance of the patient. A thorough search should be instituted to rule out a focus of infection elsewhere in the body. General measures should include a high protein low carbohydrate diet with the daily addition of one to two hundred thousand units of vitamin A. It is also advisable to eliminate foods and drugs containing iodides and bromides as well as chocolate and excesses of fats and sweets. Vaccines either autogenous or stock, are usually ineffectual.

Local measures should parallel those employed in resistant cases of *syccosis vulgaris* or lupoid *syccosis*. A satisfactory daily routine is the application of an antiseptic wet dressing for thirty minutes every morning and night followed by manual epilation of involved hairs. Satisfactory wet dressings in this disease include

1 1 2 000 bichloride of mercury in alcohol

2 Alibour water

Copper sulfate 20

Zinc sulfate 70

Camphor water q s 3000

Dilute 1 10 to 1 20

3 Liquor aluminum acetate diluted 1 5 to 1 10

4 Vlemunck's solution (1 tablespoon to 1 glass of hot water)

Subsequent to manual epilation the area should be sponged off with the remaining wet dressing solution and an antiseptic ointment should be applied

1 Compound quinolor ointment (25 to 50 per cent) containing 1 to 3 per cent sulfur

2 Antibiotic ointments (*bacitracin aureomycin penicillin terramycin*). In a disease which is so resistant to all forms of treatment a course of par-
enteral therapy (intravenous or intramuscular) with antibiotics such as streptomycin aureomycin and penicillin or chemotherapeutic agents such as the sulfonamides should also be considered.

In 1949 Larrick and Thompson successfully used streptomycin in a case of folliculitis decalvans in a 9 year-old child whose condition had resisted all other therapeutic measures. The drug was injected into the active border at forty eight hour intervals for ten consecutive treatments. Ten metaphules of streptomycin each containing 100 000 units were employed at one time by means of a hypo spray technique. There was a favorable response after the third treatment and the patient was almost cured after two months. No bad effects were reported except for minor pain in the scalp at the time of the injection.

3 Vioform (3 to 10 per cent) in petrolatum

4 Combinations of sulfur (3 to 10 per cent) and salicylic acid (3 to 5 per cent)

5 Various mercurial salves (not with sulfur wet dressings)

6 Five to ten per cent betanaphthol ointment

7 Iodine (2 to 10 per cent) in alcohol or petrolatum

8 Malachite green spray Sir Lenthal Cheatele)

These remedies are often sensitizing and irritating and must be employed under careful observation.

The use of x rays for epilating purposes has been advised in the past. It is a hazardous procedure and fraught with local and medicolegal complications. It

does not cure the disease but increases the incidence of atrophy, scarring, and resistance to subsequent measures. In select instances where the disease is confined to a comparatively small area, fractional doses of x-ray (50 to 75 r) with an approximate half-value layer of 2.0 mm Al, once weekly for four to eight weeks, may be of value.

Of merit in some cases is the frequent application of carbon dioxide slush to the papulopustular lesions. Light therapy, primarily the use of ultraviolet rays, from a source such as the water-cooled Kromayer lamp, may also be of value.

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CHAPTER IV

THE SEBORRHEIC DIATHESSES

1. THE PITYRIASES: SIMPLEX AND STEATOIDES

Clinical Features

The most common malady to involve the scalp is that which is known to the layman as dandruff, and to the physician by the misnomer, dry seborrhea (seborrhea sicca). This condition begins on the scalp as a simple, dry, scaly condition, correctly alluded to as pityriasis simplex capitis, and oftentimes develops into its immediate successor, pityriasis steatoides.

In the former, either the vertex becomes covered with loose, dry scales, or these fine, branny flakes may appear diffused uniformly over the entire scalp. This scaliness is the result of a process of continuous desquamation of the scalp, a constant flaking off of the outer layers of the epidermis. They are not due to dried out sebum, a popular misconception. The scales are gray or white and accumulate in round patches until they are shed. After they fall off the scalp, when they are disturbed by combing, brushing, or scratching, they may be found in large quantities along the hair shafts and as 'snow' on the shoulders of the persons so afflicted. Pruritus is often a subjective symptom, and the scalp may present excoriations due to scratching and acquire a reddened, thickened appearance. Normally, however, the scalp beneath the dry, furfuraceous flakes is lusterless and gray in appearance and is free from inflammatory changes.

Because of the lack of normal sebum flow, the hairs which project through these scales are dull and unruly. Due to their abnormal dryness, they are friable and therefore are frequently short and thin, with a medulla which is difficult to distinguish. When the scales are slightly greasier, they are more adherent to the scalp surface, and incline to pile up in laminated layers, matting the hair to the scalp or becoming disseminated throughout the hirsute growth.

The desquamation may spread to areas adjacent to the scalp, such as the ears, neck, forehead, and face, although this occurs more commonly in the next stage of the disorder, namely, pityriasis steatoides. Where it involves the skin in this manner, the epidermis becomes slightly reddened while the scales formed are very thin, infrequent, and more adherent than those located on the scalp.

Starting in early childhood, pityriasis simplex capitis may persist without further alteration for the entire lifetime of an individual, although a majority of persons progress into the next stage known as pityriasis steatoides. This still may be described as a non-inflammatory disturbance of a profusely exfoliating scalp, but the scaliness is more evident and the flakes become greasier and more numerous. They are decidedly more adherent, are yellowish in color, and tend to form waxy

crusts. When the scales are removed, the underlying scalp is sticky, slightly moist, and flushed instead of being grayish, and this augmented redness may extend beyond the borders of the hairy scalp, invading the forehead. In an even more advanced state Unna called these lesions the "corona seborrhea," to distinguish them from



Fig. 106—Pityriasis simplex in a young girl (Courtesy McCarthy Diseases of the Hair)



A

B

Fig. 107—A Hair covered with scales from a case of pityriasis simplex B Same hair after shampoo (Courtesy Reed Den Beste and Humoller J Soc Cosm Chem)

the corona venerea of secondary syphilis, but such lesions are actually seborrheic dermatitis, an eczematized form of pityriasis steatoides. In pityriasis steatoides, some hair fall may be observed when the sticky scales are removed because many

Thisunction may contain 3 to 5 per cent of salicylic acid for its keratoplastic and antiseptic properties, combined with 3 to 5 per cent of ammoniated mercury or the same percentage of precipitated sulfur. The best results follow if the ointment is allowed to permeate the scalp overnight and is not washed out until the following morning. Two or three times weekly, the scalp should be thoroughly massaged with a lotion containing either mercury, resorcinol, sulfur, salicylic acid, or fatty acids. These lotions are best applied with a medicine dropper in order to reach the scalp. (Detailed formulas for this entire chapter are listed in the Formulary, pages 548-550.)

In addition to these simple applications, hot oil unctions may be employed, or suberythema doses of ultraviolet light once or twice a week. Large amounts of vitamin A may also be found helpful in controlling both pityriasis simplex and pityriasis steatoides. A recent report has described the use of a selenium salt (chemically related to sulfur) in the treatment of the seborrheic diatheses. A group of cases was treated with a shampoo containing 2.5 per cent of selenium disulfide and according to these investigators, approximately 80 to 95 per cent of these cases were completely controlled by two shampoos weekly. Further studies over a longer period of time are necessary before these findings can be accepted with assurance.

2. SEBORRHEA OLEOSA

Clinical Features

The term, "seborrhea oleosa," describes an excessively oily, noninflammatory condition of the scalp and face. Other parts of the body, such as the sternal region of the chest and the skin over the spinal column may also be involved. This disorder is the result of an overproduction of sebum from the androgen-stimulated sebaceous glands. Most patients with seborrhea oleosa have had pityriasis simplex and pityriasis steatoides in early and late childhood. Their scalps, therefore, are often covered with masses of scales which at first conceal the new, oily condition beginning at puberty when the endocrine glands controlling androgen secretion begin to function. As the disease progresses, the organisms responsible for the pityriases assume an increasingly unimportant role, the scales disappear, for the most part, and only an oily secretion is left visible.

The profuse flow of sebum bathes the hair and scalp and contributes to the glossy appearance of the latter. At times the secretion is so pronounced that drops of oil form and need to be wiped off. The orifices of the glands responsible for this condition are located in the upper third of the follicles and during this imbalance of hormonal levels they become distended and filled with sebum and degenerated horny cells from the follicular linings. Pressure of the finger tips or of a glass slide easily forces the soft, grayish-white oily material to the scalp surface which, whether flushed or pallid, remains chill to the touch. Sabouraud named this mass the "seborrheic filament," and when the ducts become thoroughly plugged, this filament distends to as much as three times its normal size before forcing itself to the surface. The hypertrophy of the sebaceous glands and the profuse flow of sebum is accompanied by an overactivity of the sweat glands which misled Unna into the assumption that this disease was due to a disturbance of the latter.

The site of seborrhea oleosa is seldom confined to the scalp. The diffused oil also appears over the adjacent forehead, nose, and cheeks, absorbing particles of

crusts. When the scales are removed the underlying scalp is sticky, slightly moist, and flushed instead of being grayish and this augmented redness may extend beyond the borders of the hairy scalp, invading the forehead. In an even more advanced state Unna called these lesions the '*corona seborrhea*', to distinguish them from



Fig 106—Pityriasis simplex in a young girl (Courtesy McCarthy Diseases of the Hair)

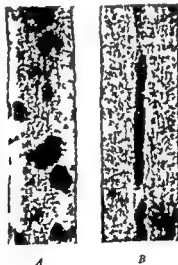


Fig 107—A Hair covered with scales from a case of pityriasis simplex B Same hair after shampoo (Courtesy Reed Den Beste and Hummoller J Soc Cosm Chem)

the *corona venerea* of secondary syphilis but such lesions are actually seborrheic dermatitis, an eczematized form of pityriasis steatoides. In pityriasis steatoides, some hair fall may be observed when the sticky scales are removed because many

hairs are found mixed in with the sticky exudation. It is perfectly possible for the patient to undergo a circular experience, suffering first from pityriasis simplex, then developing the heavier, serous laden scales of pityriasis steatoides, only, in later years, to have the serous exudation cease, the scales dry out, harden, and fall from the scalp as in the original clinical picture of pityriasis simplex.

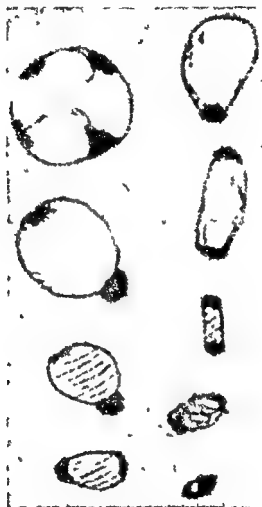


Fig. 108.—Drawing of the various forms of the *Pityrosporum ovale* of Malassez found in pityriasis simplex. (Courtesy McCarthy, *Diseases of the Hair*. Reproduced from Sabouraud, *Affections du Cuir Chevelu*, Masson et Cie.)

Pathology

Pityriasis simplex capitis begins in prepuberal childhood with the presence of Malassez's spores, which Unna later renamed the bottle bacillus. It is now known as the *Pityrosporum ovale* of Malassez, and makes its home between the desquamating scales on the scalp. It is distinctly polymorphous in appearance and may be classified under four descriptive headings. There are small spheres from 3 to 6

or 7μ in diameter, banana shaped forms, 7μ long and 2μ thick, whose ends dye more deeply with aniline than the central portion, pear shaped organisms 4 to 8μ in size, and bottle or flask shaped forms 8 to 12μ in diameter. The last named are so called because they consist of a round projection attached to a sphere and are capable of deep staining. The protoplasm is either transparent or slightly granular, and reproduction is taken care of by a yeastlike, budding process. The *Pityrosporum* never appear in chains, only in grouped colonies of ten or twelve.

Even though the epidermis suffers constant desquamation, there is rarely subsequent alopecia. Microscopic examination of the scales reveals a hyperkeratosis unrelated to inflammatory processes. Sebaceous secretion is dryer than normal, with a high percentage of cells exfoliated from the glandular orifices and follicular linings, and with imperfectly metamorphosed cells from the glands themselves.

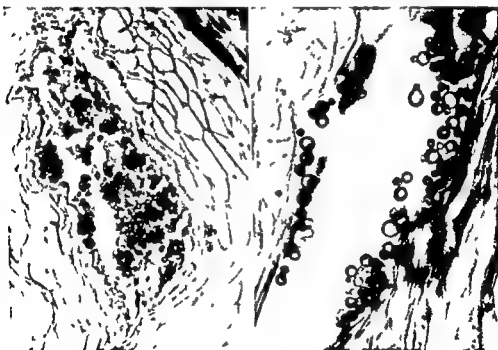


Fig. 109—*Pityrosporum ovale* in sections of the scalp. (Courtesy Sutton and Sutton. Handbook of Diseases of the Skin. Original photograph by Dr. Morris Meurer.)

The next stage manifests itself from 15 to 17 years of age following the advent of puberty. At this time the sebaceous glands become hyperactive and a mild serous exudation into the rete malpighii is observed. This serous exudate coagulates, and the leucocytes imprisoned between the layers of parakeratotic horny cells, transform the flakes into the sticky and thick scales of *pitryasis stratoidea*.

Under the microscope, stained specimens show that the spores of *Malassez* are still present in large quantities but that a polymorphous coccus (*Cedercreutz*) which Unna called a *morococcus* and which we know as a *Staphylococcus albus* has been

added. The corium is the site of a perivascular, cellular infiltration accompanied by edema and parakeratosis. It is this additional organism which is probably responsible for the serous exudate which alters the clinical picture. This coccus may be present singly, 1 to 6μ in diameter, or organisms of approximately the same size may appear in two's or four's. The morphology varies considerably. Oval forms are evident, but so are pseudobacilli, 3μ wide and 9μ long. Others are club shaped or resemble dumbbells. This coccus grows easily on Sabouraud's medium, developing a round, grayish, moist colony in forty-eight hours.

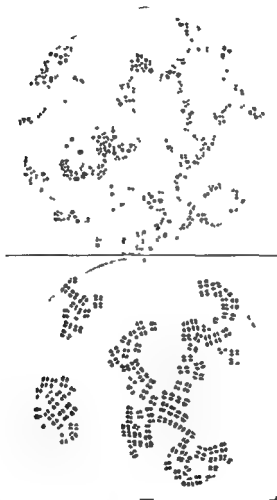


Fig. 110—Two views of the polymorphous coccus of Cedercreutz in a smear preparation from pityriasis steatoides. (Courtesy McCarthy. II sears of the Hair. Reproduced from Sabouraud. *Affect. ons du Cu r Chevelu*.)

Etiology

Barber is convinced that the *Pityrosporum* of Malassez is the organism responsible for pityriasis simplex. Pathologic studies have strengthened this belief since this is the sole organism which has been found to be actively growing on affected

scalps. The furfuraceous scales are seldom found on children younger than 11 or 12 when the androgens are just beginning to circulate through the blood stream, preparing the proper soil for the development of the *Pityrosporum*. The fact that this organism, when cultivated and inoculated in either man or beast, does not reproduce the disease, indicates that a specific soil is required for its cultivation. The periods of remission also indicate that physiologic variations in general health are conducive to its onset when these changes have the effect of altering the endocrine and nutritional balance of the scalp. Other predisposing causes include malnutrition and general debility, gastrointestinal dysfunction, intercurrent illness, blood dyscrasias, and chronic disease. Overfatigue and emotional disturbances may be factors in contributing to this predisposition to pityriasis simplex.

Just as androgens indirectly stimulate the growth of the *Pityrosporum*, estrogens inhibit its development. When administered in large enough dosage or applied locally in an ointment, the hormone successfully limits dandruff. During pregnancy, when the estrogen content of the blood rises to peak levels, sufferers from chronic pityriasis simplex often become completely relieved of this condition for the first time in their lives, only to relapse after parturition is completed.

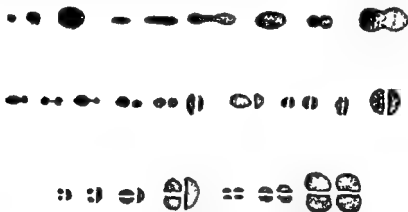


Fig 111—The polymorphous coccus of Cederereutz (Courtesy McCarthy Diseases of the Hair Reproduced from Sabouraud Affections du Cuir Chevelu)

Just as all pityriasis simplex does not develop into pityriasis steatoides, so all cases of the latter do not progress into the clinical condition known as seborrhea oleosa. The microbacillus does not develop on all scalps which have been host to the *Pityrosporum* and the *Staphylococcus albus*. It is apparently a matter of individual endocrinologic idiosyncrasy as well as bacterial infection which causes some scalps to develop one organism or two, and others to suffer from all three of the seborrheic triad.

Therapy

The scalp should be shampooed at least twice weekly. In addition, an ointment should be applied daily with a soft narrow brush in order to reach the scalp

Thisunction may contain 3 to 5 per cent of salicylic acid for its keratoplastic and antiseptic properties combined with 3 to 5 per cent of ammoniated mercury or the same percentage of precipitated sulfur. The best results follow if the ointment is allowed to permeate the scalp overnight and is not washed out until the following morning. Two or three times weekly the scalp should be thoroughly massaged with a lotion containing either mercury, resorcinol sulfur, salicylic acid, or fatty acids. These lotions are best applied with a medicine dropper in order to reach the scalp. (Detailed formulas for this entire chapter are listed in the Formulary pages 548-550.)

In addition to these simple applications hot oil unctions may be employed or suberythema doses of ultraviolet light once or twice a week. Large amounts of vitamin A may also be found helpful in controlling both pityriasis simplex and pityriasis steatoides. A recent report has described the use of a selenium salt (chemically related to sulfur) in the treatment of the seborrheic diatheses. A group of cases was treated with a shampoo containing 2.5 per cent of selenium disulfide and according to these investigators approximately 80 to 95 per cent of these cases were completely controlled by two shampoos weekly. Further studies over a longer period of time are necessary before these findings can be accepted with assurance.

2 SEBORRHEA OLEOSA

Clinical Features

The term *seborrhea oleosa* describes an excessively oily, noninflammatory condition of the scalp and face. Other parts of the body such as the sternal region of the chest and the skin over the spinal column may also be involved. This disorder is the result of an overproduction of sebum from the androgen-stimulated sebaceous glands. Most patients with *seborrhea oleosa* have had *pityriasis simplex* and *pityriasis steatoides* in early and late childhood. Their scalps, therefore, are often covered with masses of scales which at first conceal the new oily condition beginning at puberty when the endocrine glands controlling androgen secretion begin to function. As the disease progresses the organisms responsible for the *pityriases* assume an increasingly unimportant role; the scales disappear, for the most part, and only an oily secretion is left visible.

The profuse flow of sebum bathes the hair and scalp and contributes to the glossy appearance of the latter. At times the secretion is so pronounced that drops of oil form and need to be wiped off. The orifices of the glands responsible for this condition are located in the upper third of the follicles and during this imbalance of hormonal levels they become distended and filled with sebum and degenerated horny cells from the follicular linings. Pressure of the finger tips or of a glass slide easily forces the soft, grayish-white oily material to the scalp surface which, whether flushed or pallid, remains dull to the touch. Sabouraud named this mass the 'seborrheic filament' and when the ducts become thoroughly plugged this filament distends to as much as three times its normal size before forcing itself to the surface. The hypertrophy of the sebaceous glands and the profuse flow of sebum is accompanied by an overactivity of the sweat glands which misled Unna into the assumption that this disease was due to a disturbance of the latter.

The site of *seborrhea oleosa* is seldom confined to the scalp. The diffused oil also appears over the adjacent forehead, nose, and cheeks, absorbing particles of



Fig 112 —Male pattern alopecia in a 24 year-old woman with seborrhea oleosa (Courtesy McCarthy Diseases of the Hair)



Fig 113 —Male pattern alopecia in a 45 year-old woman with seborrhea oleosa (Courtesy McCarthy Diseases of the Hair)

dust and soot into the follicular orifices and causing comedo formation with, the concurrent oxidation of keratin into brown oxykeratin. Even the chest and back may be involved in the ever constant flow of sebaceous material.

Pathology

The microscope reveals that the epidermis is a nonparticipant in the pathologic picture of seborrhea oleosa. Hypertrophy and even hyperplasia overtake the sebaceous glands whose orifices are noticeably dilated because of the accelerated flow of sebum forcing its way to the surface. As Unna observed the sweat glands of the coil variety are enormously enlarged even at times forming cysts and are frequently imbedded in masses of mast and mononuclear cells such as surround the diseased follicles.



Fig. 114.—Morphology of the seborrheic bacillus. *a* Individual young forms. *b* Elements united in chains. *a* and *b* Same forms counterstained to demonstrate the capsule. (Courtesy McCarthy, Diseases of the Hair. Reproduced from Sabouraud, Affections du cuir chevelu.)

It is within these disabled follicles situated in their upper thirds that large colonies of microbacilli make their home invading the seborrheic filament. The epithelial lining of that portion of the follicle is often composed of only two or three layers of cells. Pityriasis simplex with secondary staphylococcic infection or pityriases with an underlying oily secretion (steatoides) might be confused with seborrhea oleosa. Removing the seborrheic filament with a glass slide and staining it with methylene blue makes it possible to observe both cocci and bottle bacilli in these two diseases but none of the microbacilli with which seborrhea oleosa abounds.

Ketron, in very thorough studies of the seborrheic bacillus and its cultural possibilities, discovered that it develops well in meat infusion agar to which 2 per

cent glycerin and 1 per cent dextrose have been added. The pH of the medium should be acid somewhere between 6.2 and 6.8. Should the hydrogen ion concentration of the medium be suitable for its culture, the bacillus flourishes with equal ease aerobically or anaerobically. Eventually, the growth may be characterized as luxuriant.

Sections of scalps showing long standing male pattern alopecia as well as seborrhea oleosa show that the follicles have disappeared except for a misshapen few without either hair bulb or papilla. Sclerotic connective tissue usually surrounds these remnants of the follicular apparatus. The sweat and sebaceous glands have returned to their normal size, but the blood vessels of the mid cutis are slightly dilated. All inflammatory symptoms have completely disappeared.

Light examined tissue sections of the vertex regions obtained during autopsy of patients meeting unnatural death either through accident or homicide. An apparent relationship was found between the number of hairs and the age of the individual and also the amount of infiltration of connective tissue into the fat layer surrounding the hair roots.

Etiology

We know that the endocrine constitution of each individual is one of the important factors which contributes to his propensity for male baldness or keeps him from becoming bald. The production of the male hormone in unusual amounts also gives rise in part to the seborrheic soil. It is a proved fact that androgens stimulate both the epithelium and the activity of the sebaceous glands, thus becoming responsible for the production of hyperkeratosis, seborrhea, and acne. When, in pituitary dwarfism and Simmond's disease, there is a complete lack of androgenic excitement, there is also no visible production of sebum nor its attendant seborrheic disorders. The androgens which indirectly induce hair fall are produced by the testes, the ovaries, and the adrenals. Their activity is neutralized somewhat by estrogen, but this effect can be utilized therapeutically only in very select instances.

It has long been contended that seborrhea, the "seborrheic state," and seborrheic dermatitis are causative agents and even the causative factors in the production of ordinary baldness or male pattern alopecia. In the light of modern research this theory is no longer tenable. Ordinary baldness or patterned alopecia is due in the vast majority of cases to an inherited predisposition, hormonal factors, the normal aging process and in minor degree to other physiologic mechanisms such as increased tension of scalp musculature. The association of the various seborrheic states with patterned alopecia is more than coincidental, however, because the same hormonal instigator to baldness, namely androgen, also plays a part in the development and aggravation of this group of diseases. In other words, both male pattern alopecia and seborrheic disorders may be induced and aggravated by a preponderance of male sex hormone. Once this association has occurred, it is my belief that the type and amount of hair loss may be exaggerated and increased by the seborrheic components of the process. That this association is neither fundamental nor necessary is evidenced by the many individuals with seborrheic changes who show no signs of baldness. Then too, the seborrheic soil is frequently observed in women yet baldness is comparatively uncommon. Still another fact is

the frequent appearance of seborrheic dermatitis over the occiput and in the eye brows without loss of hair in these areas. This does not mean that seborrheic dermatitis may not produce alopecia. It may not only produce alopecia but, in some instances of severe seborrheic dermatitis, alopecia of an extensive and cicatricial nature may be observed. Nevertheless it should be reiterated that the seborrheic disorders cannot be assumed to play the major role in the production of ordinary male baldness.



Fig 115

Fig 115—Cicatricial alopecia clinically similar to pseudopelade and due to a cicatrizing form of seborrheic dermatitis in a woman.



Fig 116

Fig 116—Forehead, eyelashes and nose of same patient illustrating severe degree of seborrheic dermatitis with cicatricial alopecia.

Even during the period of Sabouraud when the microbic origin was accepted without question that great clinician said that seborrhea oleosa could only develop under certain conditions of age and general health. Many years later Barber agreed that deficient exercise and imperfect nutrition could create a pathologic alteration of the skin which when combined with overactive sebaceous glands becomes so changed that it creates a perfect soil for the nourishment of the seborrheic triad of organisms. The bottle bacillus, microbacillus and *Staphylococcus albus* all flourish in this seborrheic medium. This modern conception is based on our recent understanding that vitamins as well as hormones act as biologic adjusters of equal importance both having basic chemical reactive functions as enzymic factors.

The conception that the microbacillus is primarily responsible for seborrhea oleosa is subject to the gravest doubt since normal scalps have been examined and

found to contain the microbacillus in their hair follicles without a single vestige of seborrhea. It would appear that rather than being a primary source of infection, the microorganism is actually secondary, since it flourishes only when provided with a seborrheic culture medium which, in point of time, comes first.

Other contributory etiologic factors have been widely discussed. Seborrhea oleosa may be caused by dietary excess of fats or carbohydrates, but the fault may also lie in the inadequacy of the metabolic processes rather than in their being overloaded. Thus, according to Sutton and Sutton, hypothyroidism may be a causal factor, and a trial with thyroid substance is therefore suggested by them. More recently, Jolliffe and associates have demonstrated that pyridoxine (50 to 200 mg daily, in divided doses), definitely reduces the oiliness of the skin. It seems that chemical processes which increase oxidation have a beneficial influence on the seborrheic state. Moreover, the experimental work of Perutz and co-workers has revealed that the midbrain contains an inhibitory center whose destruction is followed by excessive excretion of cutaneous fat when lipids are fed over a long period. In this connection it is particularly interesting to note that encephalitic processes are capable of causing extreme degrees of seborrhea and high grade typical comedo formation. Stokes and Sternberg call attention to the fact that the activity of the sebaceous glands is to some extent dependent upon nervous control of the secretory function. Furthermore, constipation and other abnormalities of digestion are likewise capable of increasing the seborrheic activity of the glands. The exact manner in which these disturbances exert their influence is not known. Some authorities believe that cutaneous hyperemia due to reflex mechanisms of varied etiology may constitute the underlying pathogenic basis.

Therapy

Before resorting to local therapy, the scalp must be freed of its fatty accumulations by bathing in softening solutions such as olive or almond oil and small amounts of glycerin to facilitate their removal. Even when the seborrheic masses are unusually large and adherent, soap and water will free the scales and cleanse the oily surface of the scalp. A lather of *sapo mollis* gently rubbed into the scalp and then rinsed out with water and vinegar will usually take the sticky crusts with it.

After this cleansing operation has been completed alkaline rinses may be employed to lessen the oiliness, ammonia borac, and potassium carbonate being suitable when they are carefully diluted to proportions harmless to the epidermis. An emollient application should be used immediately after these alkaline solutions to prevent possible irritation. Olive mineral and almond oils or diluted glycerin and even water may be used to bathe the scalp for this purpose. Crocker advises the use of acetic acid before the application of other lotions to aid penetration. Ointments may be substituted for oils. Petrolatum lanolin and cold cream may all be employed as the bases of bland creams with additional medicaments in low concentrations such as sulfur, resorcin, salicylic acid or the red oxide and red sulfide of mercury as well as ammoniated mercury.

Measures which might improve the state of the patient's general health are advised. Since sedentary workers are those most prone to suffer from seborrhea oleosa plenty of fresh air and exercise will prove salutary. Intelligently spaced

periods of rest the elimination of focal points of infection and a well balanced diet are also suggested. When anemia is present, folic acid iron or vitamin B₁₂ should be prescribed depending on the type of anemia. If possible, constipation should be minimized in these individuals.

When the microbacillus played a dominant role, Savill claimed great success with vaccine treatment injecting doses of from 100 to 300 million organisms, eleven times between June and October. She reported improvement in one instance after the first dose and a complete cure by December. This improvement was followed by a slight relapse due to a change in the patient's general health, but when checked seventeen years later the seborrhea had not reappeared. Barber reported the case of a patient treated for seborrhea hirsutism and alopecia with estrogen injections who benefited in two months. Another woman with male pattern baldness but neither seborrhea nor hirsutism, but with palmar hyperkeratosis responded to estradiol administered for several months.

Sulzberger and Wolf recommend the removal of the oily film by frequent shampooing with tar preparations. Oil solvents such as sulfonated oils, carbon tetrachloride, and others prove effective. These authors pointed out that while roentgen ray therapy temporarily decreases the hyperactivity of the sebaceous glands recurrences are common and more than two courses of treatment expose the patient to the dangers of excessive irradiation. Ultraviolet light, which at first seems to increase the oiliness if used biweekly over a protracted period of time may produce the desired drying effect. In the Appendix (page 549), I have listed a number of shampoos, salves and lotions of value in this disorder. It should be emphasized that frequent and adequate washing of the scalp followed by the daily application of an effective lotion and on occasion a nongreasy ointment will control the condition. Cure does not result unless the systemic measures described following the section on seborrheic dermatitis are also employed and even then, recurrences may rear their oily heads.

3 SEBORRHEIC DERMATITIS

Clinical Features

Seborrheic dermatitis is an acute subacute or chronic inflammatory disease of the skin and scalp situated on those regions most lavishly provided with over active sebaceous glands. It may present a variety of clinical features such as erythema greasy scaling exudation and crusting. Since a seborrheic soil is a prerequisite to this condition and modern writers confess that the only difference between eczema and dermatitis is etiologic the term seborrheic dermatitis becomes far more accurate than the eczema seborrheicum which Unna employed to designate this disease.

The initial lesions commonly appear on a scalp with seborrhea oleosa and seldom spread beyond its hairy confines. The greasy scaly scalp develops perifollicular erythematous macules which may become confluent during the course of the disease. When aggravated by moisture therapeutic measures or secondary bacterial invaders, these original lesions may develop into an acute, weeping eczematous dermatosis. The latter process affects the scalp far less frequently than it does the flexural aspects of the body. The yellowish red patches on the scalp vary both

in size and shape although their outlines are always irregular and sharply delineated, and they are usually covered with firmly attached, greasy, yellow scales beneath which the scalp remains moist. These scales more nearly resemble nonadherent, yellow brown crusts. A pale red or yellowish eruption may evolve in a circinate margin (Sabouraud's pityriasis circinata) below and beyond the original plaques with a tendency toward peripheral expansion. This may be noticed most clearly at the hairline of the scalp. These lesions may either remain as discrete, flat patches or merge to form sharply outlined, slightly elevated plaques with red margins and reddish yellow centers. In the more severe forms, the erythematous scalp areas become moist and are covered by thick masses of greasy, brownish scales and crusts. In some instances the expanding margins form serpiginous and polycyclic areas of a yellowish red color covered by grey, yellow, adherent scales or crusts.



Fig 117—Seborrheic dermatitis of the scalp (Courtesy Sutton and Sutton Diseases of the Skin)

In the process of extension from the scalp, or due to acute exacerbations of the disease, there is often an associated scaling and redness of the forehead, eyebrows eyelids (erythema blepharitis) nasolabial folds, beard, ears (external meatus and postauricular area) and neck. Although the scaling forms are the most common and constant manifestations of seborrheic dermatitis on the scalp there is no unanimity of the process and dry forms may slowly or suddenly become moist and vice versa. In children the process may begin as one or several small patches in the scalp with gradual spread over the entire scalp surface. A complicating strep

to coccal infection occurs more frequently in children than in adults, converting the process into a superficial pyoderma characterized by bright red erythema, exudation, impetiginous crusting, and fissuring. The ears and postauricular regions are most frequently involved by this infectious, eczematoid process.



Fig. 118—Seborrheic dermatitis of the axilla. (Courtesy Sutton and Sutton. *Diseases of the Skin*.)

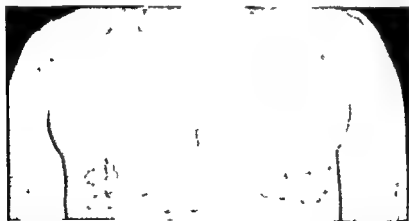


Fig. 119—Seborrheic dermatitis of the psoriasisform type with typical involvement of sternal area. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph by Dr. F. Ronchese.)

It is only within the scope of this book to discuss seborrheic dermatitis in so far as it involves the scalp, although brief allusions to its manifestations elsewhere on the body surface are included. The reader is referred to the general textbooks

on dermatology for a complete description of the disease as it affects the glabrous skin. In general, it may be stated that the clinical appearance of seborrheic dermatitis is dependent on the habitus of the individual. It may range from a mild type exemplified by a greasy, yellowish, scaling erythema of the scalp, eyebrows, nasolabial folds and sternum, to a severe form characterized by exudative, eczematous, and fissured plaques in the postauricular, axillary, umbilical, and anogenital regions. In the latter instances, a superimposed moniliasis is not uncommon. Areas other than the scalp may also be involved by pityriasisform, papular, and flower leaf or petaloid types of seborrheic dermatitis and may pose very difficult problems in differential diagnosis.

Differential Diagnosis

Seborrheic dermatitis may be distinguished from psoriasis because of the greasy character of the former's scales, the yellowish tint which invariably accompanies the underlying erythema, the lack of capillary bleeding after scale removal, and the general course of the eruption. The seborrheic corona is a sharply defined, red scaly ribbonlike band usually observed at the hair margins as in psoriasis but unlike the latter, it has an even, regular outline and is a far more superficial process. In the initial stages of psoriasis, McCarthy found differential diagnosis somewhat baffling but as soon as the scales formed they assumed a silvery sheen rather than the greasy yellowish tint of seborrheic dermatitis, they were thicker, and could usually be found on the elbows, knees, and other parts of the body typically affected in psoriasis.



Fig. 120—Seborrheic dermatitis in children. Note the resemblance to psoriasis. (Courtesy Dr. E. Urbach. *Skin Diseases: Nutrition and Metabolism*. Grune & Stratton.)

The variation of this disorder sometimes referred to as seborrhuasis shows components of both diseases and is often extremely difficult to differentiate clinically. Differential diagnosis may be histologically verified. Between the lamellae of nucleated horny cells there are dense concentrations of leucocytes and dried serum.

with parakeratosis either covering the entire lesion or isolated sections. Acanthosis is marked and the tips of the extended papillae approach the parakeratotic layer but not as closely as in psoriasis. Partial mononuclear infiltration may be noted in the papillary bodies in seborrheic dermatitis, but in psoriasis the infiltration is polymorphic. Should the scales be removed and the lesions touched lightly with an absorbent tissue, the presence of psoriasis would be suggested by the appearance of discrete blood puncta, while seborrheic dermatitis would stain the tissue with oily droplets.

Simple dermatitis or eczema does not often originate on the scalp. Clinically, they lack the oily secretions and heavy crusts of seborrheic dermatitis and are wanting in the yellowish tint and the sharply delineated patches. In addition these diseases do not spread peripherally in circinate outlines and they usually result in more severe subjective symptoms than those suffered by patients with seborrheic dermatitis. A careful history may help elicit the specific causative agent.

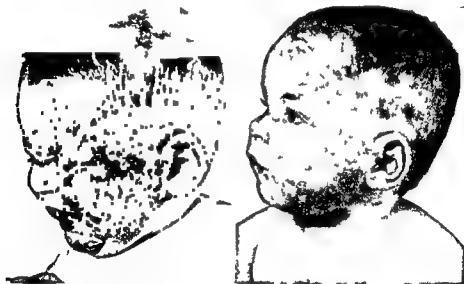


Fig 121—Infantile eczema (Courtesy Dr H Urbach *Skin Diseases, Nutrition and Metabolism*)

There is little difficulty in distinguishing this disease from lupus erythematosus because the scales produced by the latter are dry and so adherent that they cannot be removed by lifting but must be scraped off. Other distinguishing features of lupus erythematosus include telangiectasia, follicular plugging, atrophy, and the presence of typical lesions on the face and ears. Likewise, therapy helpful in seborrheic dermatitis is totally ineffectual in lupus erythematosus.

A chronic streptococcal infection of the scalp and retroauricular folds may present a picture similar to that of seborrheic dermatitis but the scales are not as thick, they are white rather than yellow in color, fissures form in the depths of the folds and exhibit small pink, oozing points after encrustation and its removal. The presence of a pyogenic streptococcus rather than a bottle bacillus in scrapings from the reddened tissue would help prove the diagnosis of the disease under discussion.



Fig 122—Seborrheic dermatitis ("cradle cap") (Courtesy Dr E Urbach Skin Diseases, Nutrition and Metabolism)



Fig 123—Seborrheic dermatitis, extreme degree in neglected scalp (Courtesy Dr F Combes)

The differentiation between seborrheic dermatitis and infantile dermatitis is often very difficult to establish. However, in patients of this age, seborrheic dermatitis is likely to begin with intertrigo in the groin, axillae, and other folds. Furthermore, blepharitis is very commonly present. Another characteristic symptom is the greasy scaling of the scalp ('cradle cap'), which may extend to other parts of the body, usually to the face, neck, shoulders, and trunk, forming large areas of irregular configuration by fusion. The eruption is essentially a dry, scaly one, usually with rather sharply defined margins, and of a yellowish pink color. The patches do not ooze unless they are rubbed. In severe cases, erythroderma may develop, its severest expression being Leiner's disease (erythroderma desquamativum). The characteristic primary lesion is a red, scaly papule which later is transformed into a greasy, scaly crust. Infantile dermatitis, on the other hand, is characterized by intense itching, exudation, crust formation, and secondary infection, furthermore, the skin folds are not involved, and there is no blepharitis.

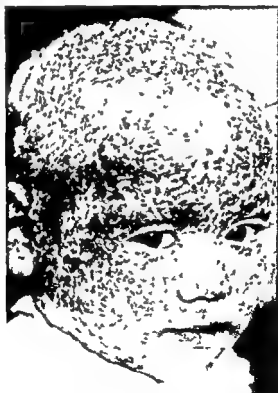


FIG. 124.—Infantile eczema. (Courtesy Sutton and Sutton. *Diseases of the Skin*.)

Occasionally, the macular types of seborrheic dermatitis may resemble the lesions of secondary syphilis. However the seborrheic lesions show a predilection for the scalp, nose, cheeks, postauricular, presternal and scapular regions, whereas macular syphilis is rarely seen on the face or anterior chest region. The fine, yellowish, greasy scale is unlike the pink nonscaling macule of syphilis. The latter lesion remains discrete, whereas the seborrheic lesions tend to become confluent, especially

in the flexures and body folds. Finally, the acute seborrheic eruption may be eczematous and even vesicular in contradistinction to the macular syphilide, which is never vesicular, exudative, or crusted. The diagnosis is actually difficult only when a macular secondary syphilide is superimposed on a seborrheic soil and assumes the characteristics of seborrheic dermatitis ("the seborrheic mask of secondary syphilis"). Occasionally seborrheic lesions assume a circinate form which has been described as seborrhea petaloides and resembles a late syphilide. From the features described above, differentiation between the two may be simplified.

Pityriasis rosea may easily be confused with the scaly, macular, oval type of seborrheic lesion occasionally observed on the trunk and extremities. Where only a few lesions are present, the diagnosis may be extremely difficult. However, in the usual case, the differentiation is clarified by the presence of the typical involvement of the scalp by diffuse, erythematous patches covered with a greasy, yellowish scale with concomitant involvement of the eyelashes, nasolabial folds and other seborrheic areas of predilection. Pityriasis rosea may involve the scalp, especially in children, but in that location the sparse lesions are usually discrete, erythematous macules covered by a fine dry scale and rarely persist more than six to eight weeks.

According to Hill, the "seborrheic" infant with eczematous involvement of the cheeks and scalp is likely to respond to dietary measures. He found that of 126 milk sensitive, eczematous infants, 104 had the onset of this eruption before the age of 6 months and were helped by simple boiling of the milk (lactalbumin sensitive). Gordon divided "scurfy scalp" in infants into three types: (1) "milk crust" due to infrequent cleansing with soap and water, (2) true dandruff, acquired from the mother, (3) mixed types. Mildly stated, this is descriptive oversimplification.

Pathology

Under the microscope it becomes evident that the invasion of the epidermis by the triad of seborrheic organisms multiplies the outermost layer of cells, causing the stratum corneum to form scales. Stained with methylene blue, clusters of large, flask-shaped, ovoid and rounded bodies may be located lying on and between the layers of laminated epidermal cells. These prove to be the *Pityrosporum* of Malassez. Moore, Nile, and the Engmans produced lesions histologically similar to seborrheic dermatitis with cultures of *Pityrosporum ovale*. Benham in a study of this *Cryptococcus*, believed it to be a lipophilic fungus capable of culture, although with difficulty, on wort agar soaked in oleic acid and other fatty substances. Examination of the scales from severe cases also reveals chains of the *albus* strain of *Staphylococcus* in the vesicles of the stratum corneum. Merrill, investigating 21 cases of seborrheic dermatitis found two varieties of diplococci and a bacillus. One of these cocci was chromogenic, possibly a cause of the yellowish color which characterizes this disease.

In the rete malpighii under the areas of parakeratosis there is both intercellular and intracellular edema, a spongiosis with an inflammatory cellular infiltrate, a slight acanthosis, a swelling of the papillae only barely discernible in the middle layer of the corneum, and, lastly, lymphocytes and leucocytes within the dilated vessels of the midcorium. The tissue reaction is far more severe in the unusual case showing cicatricial alopecia.

Etiology

The underlying cause of seborrheic dermatitis is presumably pityriasis steatoides and seborrhea oleosa. The excessive flow of sebum on the surface of the skin in turn depends upon an overactivity of the androgen producing glands of internal secretion. Ordinary seborrhea oleosa develops into seborrheic dermatitis when the inflammatory changes become apparent. The chemical decomposition of unusual quantities of sebum on an unclean surface of the skin may suffice to encourage such a transformation. The site of the diseased epidermis has become sensitized to rather than infected by, microorganisms with vesicle formation followed by oozing and crusting. MacLeod and Dowling, using a *Pityrosporum* culture inoculated typical seborrheic skins and produced characteristic eruptions of seborrheic dermatitis. The same strength culture inoculated on normal skins caused only the temporary development of follicular papules.

Recent studies of the flora of normal skins have revealed that the bottle bacillus *Staphylococcus albus* and microbacillus are all present though in small quantities and presumably inactive. It is when the sebaceous gland secretion is chemically changed and increased in volume that these organisms begin to thrive and become transformed from inactive saprophytes to active parasites with pathogenetic effects. Barber believes that the hormonal factor is the most important excitant. Not only do androgens stimulate keratinization of the skin and pilosebaceous follicles and overactivate the secretory process of the sebaceous glands but they seem to encourage the growth of the three seborrheic organisms mentioned above. In the occasional patient the administration of estrogens has been of value (see page 101).

Although these facts suggest the underlying causal factor to be an androgen-estrogen imbalance, Andrews, Post and Domonkos state that seborrheic dermatitis is essentially a nutritional rather than a hormonal disease and that the microorganisms are secondary invaders responsible for the inflammatory symptoms. Individuals suffering from this disease are adversely affected by diets which encourage the retention of fluids within the tissues and are rich in alcohol, fats and carbohydrates and deficient in proteins and the vitamin B complex.

There are also a variety of other etiologic possibilities including hypothyroidism, hereditary factors, chronic gastrointestinal disorders, inadequate personal hygiene, psychogenic factors, lack of exercise and ordinary fatigue. All these may in variously proportioned combinations contribute to the seborrheic soil necessary for the culture of organisms associated with seborrheic dermatitis. Locally excessive heat, moisture and friction may hasten the onset and both aid and abet the seborrheic state.

The relationship between the seborrheic d adtheses and male pattern alopecia has been adequately discussed elsewhere (pages 54, 182 and 229). In general it may be said that although the seborrheic disorders do not play a primary role in male pattern alopecia they may still play an as yet undetermined supporting role as far as aggravation of the process is concerned. Furthermore, long standing and severe cases of seborrheic dermatitis with recurrent bouts of secondary infection may give rise to a clinical picture resembling folliculitis decalvans. In these uncommon instances the end stage is that of an extensive cicatricial alopecia of the scalp commonly referred to in the literature as pseudopelade.



Fig 125 —Cicatrizing seborrheic dermatitis clinically simulating folliculitis decalvans (Courtesy Dr Carl Laymon and J Invest Dermat)



Fig 126 —Histologic section from scalp of patient with cicatrizing seborrheic dermatitis. The tissue shows pyogenic crusting, acanthosis and edema of the epidermis, and moderately intense inflammation of the cutis (Courtesy Dr Carl Laymon and J Invest Dermat)

Therapy

Those patients suffering from the seborrheic diathesis often complain of blepharitis gastrointestinal disorders, psychogenic disturbances, and constipation. These difficulties are enhanced by their interference with the normal utilization of vitamins. The underlying digestive imbalance negates even a more than adequate vitamin intake. This may be helped by proper diet and the administration of vitamin B complex combined with crude liver extract.

Vitamin B₁₂ and its concentrates were employed therapeutically by Andrews in one hundred dermatologic patients. The dose of vitamin B₁₂ was 10 to 30 μ g once a week or as infrequently as once every two or three weeks, administered intramuscularly. Many of those suffering from seborrheic dermatitis have a slight normocytic or hypochromic anemia which could not, however, be used in determining the response of the skin disease to this therapeutic measure. Neither a red blood cell count, hemoglobin or hematocrit studies, nor reticulocyte counts were indicative of the progress made. The only check was constant observation of the changing clinical features and the patients' own impressions of their well being. Local treatment with antiseborrheic preparations containing the usual combinations of sulfur, resorcinol, oil of cade, salicylic acid or ammoniated mercury was limited as much as possible to make certain of the value of the injections. Andrews stated that vitamin B₁₂ is not a specific for seborrheic dermatitis as it is for pernicious anemia but is used only in conjunction with other therapeutic measures, requiring a much more concentrated dose to be effective than in cases of anemia. His patients with evidence of widespread secondary staphylococcal infection were also given penicillin or sulfonamides. After two or three treatments with vitamin B₁₂, immediate and spectacular results were obtained. The condition was cleared and recurrences were checked by maintenance doses of 15 or 30 μ g of the vitamin injected intramuscularly every two or three weeks. There was no reappearance of symptoms when all metabolic and nutritional disorders and focal points of infection were corrected. Sixty eight per cent of those studied had seborrheic dermatitis located on the scalp. Of the 100 cases only 37 were thoroughly studied. Of these 43.5 per cent were apparently cured or greatly improved and the same percentage were improved to some degree. Several of the patients successfully treated in this group had histories of innumerable treatments involving the use of local applications, injections of thiamin, applications of x ray and doses of penicillin. I have been unable to confirm these observations of remarkable improvement with vitamin B₁₂ therapy in my own patients and believe it to be of questionable benefit.

No matter what therapeutic measures are chosen, all patients should be advised to follow a daily regimen of regular periods for rest and sleeping, frequent bathing, the use of soap substitutes if soap should prove to be an irritant, a diet high in protein and low in fats and carbohydrates, the abolition of alcohol and as much fresh air and exercise as possible.

In the acute and exudative stages of seborrheic dermatitis, wet dressings are of value, especially when impetiginous and severe inflammatory changes are present. The usual wet dressings employed are aluminum acetate (5 per cent), boric acid (5 per cent), sodium propionate (5 to 10 per cent), and weak antiseptic solutions.

As the acute stage subsides, a mild ointment or lotion may be prescribed. The manner of application is of great importance in order to insure thorough coverage of the affected site. This technique has been described elsewhere and may be restated briefly by emphasizing that the local applications should be applied to repetitive partings in the scalp. The most useful preparations contain sulfur, salicylic acid, mercury (not in combination with sulfur), vioform, resorcinol monoacetate, and the various tar fractions in amounts of 1 to 5 per cent. Typical formulas may be found in the Appendix, page 549. If infection is still present, it is useful to add or substitute bacitracin, terramycin, aureomycin, or other antibiotics to the preceding formulas. It must be recognized that any of these preparations are capable of acting as irritants or sensitizing agents, and should be carefully watched. Aggravation of the local process may necessitate temporary or permanent discontinuance of a particular remedy.

In my experience, chronic seborrheic dermatitis may prove to be resistant to all forms of therapy. In such cases, a cure can only be effected with the complete cooperation of the patient. General measures such as moderate exercise, adequate sleep and rest, and proper diet are fundamental. The diet is basically a low fat, low carbohydrate, high protein type, with the elimination of alcohol and excessively hot foods and drinks. In the obese, plethoric individual, it is advisable to make certain that the patient does not indulge himself with the mundane pleasures of steam cabinets, Turkish baths, and daily massage, as these activities all tend to increase the seborrheic diathesis. (Endocrine therapy has been discussed elsewhere in this volume.) The emotional components of the process as evidenced in part by the labile vasomotor 'flush' reactions require guidance, reassurance, and oftentimes readjustment. Wittkower and MacKenna observed that persons with seborrheic dermatitis are more likely to be grossly inhibited in social contacts, are conscientious, worrisome, anxious, and unable to relax. I believe that patients with chronic seborrheic dermatitis are never cured unless proper attention is paid to the interrelationships of psyche and soma (see page 384).

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CHAPTER V

INFECTIONS OF THE SCALP

INTRODUCTION

The scalp is a common site for infectious disease. Almost any type of pyogenic bacteria, virus, fungus, or other organism may produce a more or less characteristic picture on this portion of the anatomy. Although the disturbance may be similar to one produced elsewhere on the body surface by the same infecting agent, it is always modified and conditioned by the physiologic and morphologic features peculiar to the scalp.

I BACTERIAL INFECTIONS

1 IMPETIGO CONTAGIOSA

This contagious disease of the skin is a superficial staphylococcus or streptococcus infection. The lesions are discrete, thin walled vesicles and bullae which rapidly become pustular and then dry to form loosely adherent, honey colored "stuck on" crusts. This disease usually occurs in children and the face is the site of predilection. The eruption often takes its origin near the nostrils, mouth, and eyelids. The scalp, especially the occipital region, is a not uncommon site, especially in children having pediculosis capitis. Once the scalp becomes involved, improper treatment may result in spread throughout the entire hairy area. Where the lesions are recent and active they appear as circinate, erythematous, lightly crusted lesions. Not uncommonly, the scalp also shows a layered arrangement of crusts due to the spread and overlapping of series of these lesions. The disease may pass unnoticed in the scalp until the crusts dry and fall off, pulling the matted hairs with them. This resultant alopecia may be the first sign of scalp involvement. Depending upon the depth of this involvement, impetigo of the scalp may lead to temporary alopecia, with small round or oval hairless patches. If the infection has been sufficiently severe or deep, this alopecia may terminate as permanent cicatricial patches.

In the severe and extensive forms of impetigo neonatorum also, the scalp may be involved.

2 IMPETIGO OF BOCKHART

(SUPERFICIAL PUSTULAR PERIFOLLICULITIS)

Clinical Features

This disease is a staphylococcal perifolliculitis in which small pustules surround the orifices of the pilosebaceous glands, the favorite locations being the extremities and the scalp. The disease differs from impetigo contagiosa in that the

lesions develop in the ostia of the hair follicles and extend downward to create a definite folliculitis. The initial lesions are primarily pustules containing a light yellow pus often centered by a hair and surrounded by an inflammatory areola.



Fig. 127—*Impetigo contagiosa*. (Courtesy McCarthy. Diseases of the Hair.)



Fig. 128—*Impetigo neonatorum*. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Dr. S. Swetzer.)

The pus collects under the horny layer, elevating and distending it. The lesion varies in size from 1.5 to 6 mm. Although the scalp is a common site of black hair's impetigo, the pustules are not infrequently very numerous and may spread to involve many parts of the body. Some of the conditions favorable to its de-

velopment include preceding infections, dirt, friction, pruritus, scabies, and various superficial forms of dermatitis (following the use of ointments such as the mercurials and tars). On the scalp, the pustules are frequently very small, almost pinhead in size, surrounding the hair shaft. Such lesions rapidly spread through the scalp of infants and young children due to itching and subsequent uncontrolled scratching and rubbing. Under the name of *folliculitis de la forme aigue militaire*, Sabouraud described a rare type of this form of impetigo. The eruption is of sudden onset, the entire scalp becoming erythematous and sensitive and rapidly covered or studded with innumerable minute superficial pustules. This type of eruption is associated with an extreme degree of itching, and the subsequent scratching is in part responsible for its rapid spread. According to McCarthy, it is more common in young blond adults between the ages of 20 and 30 years, especially following periods of dietary indiscretion, constipation, and excessive alcoholic indulgence.



Fig. 129.—Impetigo of Bockhart. The characteristic pustules have dried out to form perifollicular crusts. (Courtesy McCarthy, *Diseases of the Hair*.)

The walls of the pustules of the Bockhart type are usually tougher and more resistant than those of impetigo contagiosa. This is a partial explanation of the fact that they persist for a longer term without rupture and are resistant to many forms of therapy. In some instances the disease is accompanied by fever and swelling of the regional lymph nodes. It is not uncommon to see an associated number of small patchy areas of temporary alopecia. Occasionally, Bockhart's impetigo of the scalp may occur as a complication of infectious eczematoid dermatitis of the ears.

Pathology

Histologically, the lesions of impetigo primarily affect the epidermis. The individual lesions are subcorneal or intracorneal vesicles or bullae. These lesions

may be filled in part or entirely with polymorphonuclear leucocytes fibrin, small round cells, and the infecting organism. In the Bockhart type, the lesions primarily involve the follicular ostia. Otherwise there is very little change in the epidermis. The corium shows a mild superficial dilatation of the blood vessels, with a perivascular infiltration composed mainly of small round cells and polymorphonuclear leucocytes.

Etiology

The causative agents of the various forms of impetigo are either staphylococci or streptococci. The Bockhart type is usually of staphylococcal origin.

Therapy

Impetiginous processes involving the scalp are difficult to treat because of the matting together of the hair by crusts and cellular debris. A fundamental objective is the removal of all crusts in order to bring the effective agents to the base of the lesions. This may be accomplished within a few days by means of antiseptic and astringent wet dressings such as Burow's solution (1 to 10) alibour water (1 to 10) and mechanical softening of the crusts by the application of soap and water. Unless there is a very considerable degree of inflammation the frequent use of soap and water is of great value in the treatment of impetigo of the scalp. In some instances the use of dilute solutions of hydrogen peroxide or boric acid solution are also efficacious in softening the crusts. Where the hair is thickly matted and crusted together it may be necessary to shave the entire scalp or small portions of it. When many pustular lesions are present on the scalp it is necessary to open these lesions under strict aseptic precautions before applying wet dressings and subsequent bactericidal agents.

In my experience the most effective local remedy for pustular processes involving the scalp is a member of the quinoline family. In the usual case the initial preparation employed is a 25 or 50 per cent concentration of Quinolol Compound Ointment containing 1 to 2 per cent of sulfur. This is gently massaged into the scalp morning and night. Other members of the quinoline family such as Vioform Sterosan and diodoquin are also of value. Occasional instances of sensitivity to these drugs do occur but they are less frequent than those encountered during the usage of the classical remedy for impetigo ammoniated mercury. Although the latter remedy in a 5 per cent concentration appears in the literature to be synonymous with proper therapy for impetigo in its various forms there are many more effective and less irritating preparations for the scalp available at the present time. Any or all of the chemotherapeutic and antibiotic agents are effective in the treatment of the impetiginous processes namely sulfonamides penicillin aureomycin streptomycin chloramphenicol and terramycin. Although these drugs are effective from the standpoint of both topical and systemic administration they should be reserved for extremely resistant and extensive types of this disease. The reason for this statement is an obvious one. Their use for some local disease of trivial nature may result in sensitization of the individual to these drugs thereby precluding its usage for some future and more serious ailment. However bacitracin is effective locally and as yet is but little used for systemic disease.

In some instances, ointments are not well tolerated on the scalp and it is necessary to restrict local applications to the various wet dressings and both aqueous and alcoholic solutions of bactericidal remedies such as those mentioned previously. Some of the older remedies are also of value in aqueous or alcoholic extracts and these include iodine (1 to 3 per cent), thymol (1 to 2 per cent), silver nitrate (0.25 to 5 per cent), and resorcinol (3 to 5 per cent). Intraderm Tyrothricin Solution may also be employed. When the scalp shows an extensive inflammatory reaction as in eczematous dermatitis, it is advisable to restrict all measures to the use of soothing wet dressings. In these severe cases, it may also be advisable to apply one of the more potent chemotherapeutic agents via the parenteral route. Where impetiginous involvement of the scalp is secondary to other processes (pediculosis capitis, infectious eczematoid dermatitis), therapy must be directed toward eradication of the underlying causative agent or focus of infection.

3 FURUNCULOSIS AND CARBUNCULOSIS

Clinical Features

A furuncle is a staphylococcal infection starting in the hair follicle, about which a local cellulitis develops, with eventual central necrosis. A succession of such lesions constitutes furunculosis. Although these lesions may occur anywhere on the body surface, the back of the male scalp is a not uncommon location. Initially, the lesion appears as a red, tender, slightly indurated nodule aggravated by friction from the collar. In a short period of time it increases in size and the neighboring scalp tissue, though not inflamed, becomes edematous, hot, and sensitive. As pus forms, the center softens and a yellow midpoint becomes apparent. This abscess finally ruptures and a light yellow or blood tinged pus escapes. After a day or two, a necrotic central plug may also be discharged. Subsequent healing is eventually followed by a small patch of cicatricial alopecia. In many instances a succession of furuncles may occur in the same location over a period of months and even years.

A carbuncle is similar to a furuncle but occupies a larger area of skin and subcutaneous tissue. The back of the neck is also a frequent site for the carbuncle, which may start as a furuncle and spread rapidly through the adjoining skin and subcutaneous tissue. Often the disease is accompanied by systemic symptoms such as fever, chills, and malaise. The lesion at first resembles a furuncle in that it is a hard, red, sensitive, indurated mass, but of larger size. Gradually softening takes place and pus exudes, not from one but several apertures. The involved area becomes undermined and the entire mass is cribriform, inflamed and fluctuant. The lesion is frequently covered by a large slough which is subsequently discharged in one piece. Healing is gradual and eventuates in a large, smooth bald patch. Although any part of the body may be involved, the disease is most commonly encountered at the nape of the neck.

Pathology

Histologically a furuncle shows a massive folliculitis with necrosis of the perifollicular cellular tissue. In a carbuncle the lesions are more extensive and the necrosis of the perifollicular cellular tissue becomes confluent over large areas. The dif-



Fig 130—Patchy alopecia of a permanent nature resulting from a recurrent perifolliculitis and pyoderma of the scalp over a period of eleven years. Note the bizarre and irregular outlines and the small size of the patches. (Courtesy McCarthy: *Diseases of the Hair*.)



Fig 131—Temporary alopecia following a localized patch of superficial folliculitis. Secondary dermatitis due to ammoniated mercury ointment is also in evidence. (Courtesy McCarthy: *Diseases of the Hair*.)

ference between a carbuncle and furuncle is somewhat more than a matter of degree. In a furuncle, the inflammation starts in a follicle and always remains definitely circumscribed with the follicle as its center, although it may extend as far down as the hypoderm. In a carbuncle, the infection extends into the hypoderm and then spreads laterally and extends to the surface where it appears as a group of secondary infections in the vicinity of the primary lesion. The danger of phlegmonous formation and rapid spread under the *galea aponeurotica* is ever present. The openings of a carbuncle are accordingly multiple, irregular, and winding.

Etiology

The causative organism of furunculosis and carbunculosis is the staphylococcus. The hair follicle and adjacent sebaceous glands are also infected, the inflammation involving the immediate connective tissue. Predisposing causes include both cutaneous and systemic diabetes, obesity, blood dyscrasias, poor hygiene, irritation and friction from clothing (especially tight fitting collars), drugs (iodides and bromides especially), and lowered resistance due to systemic disease.



Fig 132 —Carbuncle showing typical location and central slough (Courtesy Sutton and Sutton Diseases of the Skin)

Therapy

Therapy depends upon the acuteness of the process. In the early stages of a furuncle or carbuncle involving the scalp or nape of the neck, when the lesion is merely a tender, red elevation, the lesion may often be aborted by a combination of x-ray therapy and chemotherapy. At this stage, a small amount of x-ray therapy applied directly to the lesion may result in rapid absorption of the entire process. I employ a dosage of 35 to 50 r with a low voltage technique (60 kv) administered daily for two to three days. In addition, the patient is advised to apply hot, wet dressings of Burrow's solution 1 to 10, or 10 per cent boric acid solution every three to four hours for thirty minutes. If treated early enough, this combination alone is adequate although the employment of chemotherapy will considerably in-

crease the chances for a speedy recovery. Adequate therapeutic levels may be reached by the administration of 300 000 units of penicillin daily for several days, terramycin or aureomycin given in divided doses for a total of 1 to 2 Gm daily for several days or sulfathiazole in doses of 2 to 3 Gm daily. Antibiotics such as penicillin and bacitracin may also be injected directly into the lesion.

Where the process has already progressed to central pustulation or discharge, local measures should consist of hot wet dressings every few hours for 20 to 30 minutes. The usual solutions employed are Burow's 1 to 10, boric acid 5 to 10 per cent, alibour water 1 to 10, Vlemminckx's solution 1 to 10, and magnesium sulfate 1 to 5. Between wet dressings the scalp should be covered with a layer of bacitracin ointment. The latter has been found very satisfactory due to its low incidence of sensitization, although comparable results may be secured by the local use of 5 to 10 per cent ammoniated mercury ointment, aureomycin or terramycin ointment, penicillin ointment and 25 to 50 per cent Quinolor Compound Ointment. The latter preparations may prove locally irritating. More rapid absorption of the process may also be hastened by the use of chemotherapeutic agents in aqueous solution for local irrigation and instillation. Aqueous solutions of bacitracin or penicillin may be injected directly into the lesion and surrounding skin employing any of several techniques reported in the literature. In the more advanced lesions, oral and/or parenteral penicillin, terramycin, aureomycin and other chemotherapeutic agents are administered concurrently with the local measures. Surgical incision and drainage are employed far less frequently than in former years although the occasional extensive carbuncular process in the scalp may require surgical drainage in addition to the measures described. This is especially true in those instances where rapidly spreading deeply seated infections may undermine large portions of the scalp and both drainage and high dosage chemotherapy are urgently indicated. In the scalp it is particularly important to observe the utmost precautions as far as cleanliness and local hygiene are concerned once an infection has commenced. The obvious reason is that local spread easily occurs because of the numerous openings of the adjoining follicular orifices. Accordingly, once an infection has made its appearance the adjoining scalp must be kept scrupulously clean by frequent washing with an antiseptic soap solution preferably one containing hexachlorophene. These washings should be performed several times a day in addition to the application of local wet dressings and antiseptic ointments.

Pustular infections of the types mentioned are often recurrent problems in the scalp. In such instances reinfection may be minimized by proper local hygienic measures. Frequent washing of the scalp with an antiseptic soap and the daily application of a lotion containing 0.1 per cent bichloride of mercury or 1 to 2 per cent hexachlorophene are of value. As in recurrent infectious processes elsewhere on the body, the individual should be studied to rule out the presence of an underlying systemic disease such as diabetes, an unsuspected focus of infection or blood dyscrasia. In the majority of instances a low carbohydrate and low fat diet, with the avoidance of chocolate, shellfish, drugs such as iodides and bromides and highly seasoned or greasy foods is of value. Specific immunologic procedures are usually of no great aid in minimizing recurrences although the author has seen occasional

instances of cure following the use of autogenous vaccines or stock toxoid preparations. Such instances are definitely in the minority. Proper local hygiene and rational dietary control appear to offer the major points of resistance to recurrence.

4 LUPOID SYCOSIS

Clinical Features

The first lesions of lupoid sycosis usually appear on the bearded area of the face, on the upper lip around the chin, or anterior to the ear. Where the temple is affected, as it often is, and the site of the disease is enlarged by centrifugal extension, the neighboring scalp may become involved. Galewsky found that seven cases of the nine he treated were unilateral, the minority showing symmetrical plaques. When the scalp has once been invaded the lupoid sycosis may either border the entire area, leaving only the top of the head free, or it may extend partially around the scalp in a snakelike margin several centimeters wide. In spite of its comparable location, it is impossible for this disease to be confused with marginal alopecia areata because of the presence of folliculitis, erythema and, eventually, cicatricial tissue.

An erythematous inflammatory patch is often the first indication of the disease. Scattered vesicles and pustules then appear about the hairs, and the patch begins to exude a light colored serous fluid. Exudation is followed by the formation of thick crusts. With increased inflammation, there appear large, perifollicular pustules pierced by hairs. This stage may be defined as discrete, pustular folliculitis because, while the areas affected tend to coalesce as the disease spreads peripherally, the pustules themselves do not. During the desquamative period, there is moderate itching and burning.

A few days after the onset of the disease, it is possible to remove the involved hairs with only the slightest traction or, if left to themselves these will loosen and fall. The roots of the hairs are tumid and sodden, encased in a sheath composed of the ectodermal wall of the lowest section of the follicle. After the hair is gone, either having been epilated or fallen of its own volition, the inflammation decreases. In the final stage, the skin pales and becomes the white, glossy, depressed plaque which we associate with permanent alopecia characterized by complete atrophy of the pilosebaceous system.

During the course of the disease, the center of the involved area exhibits a keloidlike scarring following the original deep seated abscesses. At the same time, it is bordered by a zone 1 to 2 cm. wide which is still composed of active brown and deep red papules or pustules. New papules appear as old ones involute and in this way the disease may persist for years.

Originally, lupoid sycosis was thought of as a more extensive inflammatory version of sycosis vulgaris with an unusual amount of infiltration of the derma but there are actually several points of difference. The deep pustules which undergo a colloidal degeneration Brocq noted had a close resemblance to the apple jelly nodules of lupus vulgaris and it was this which caused him in 1888 to name this type of sycosis 'lupoid'. Also in this type the lesions occur only in patches, while

in sycosis vulgaris there is usually a total regional involvement. It differs further because of its protracted course and even more stubborn resistance to therapy which may be beneficial for sycosis vulgaris.

There are actually two kinds of lupoid sycosis. There is a variety which does not develop posterythematous folliculitis and perifolliculitis but the infiltrate results in the same type of cicatricial alopecia. The toxic action of the infecting agent must be equally as potent in these cases because the atrophy is equally complete. Unna called this *ulerythema sycosiforme* defining his term *ulerythema* as a pathologic process which results in atrophic scars without suppuration or ulceration but chiefly through the absorption of a cellular infiltrate.



Fig. 133.—Lupoid sycosis showing cicatricial alopecia and active pustular lesions in the margins. (Courtesy Dr. Marion Sulzberger.)

Except for the very last stage during which lupoid sycosis resembles pseudo pelade there is little difficulty with differential diagnosis. Lupus erythematosus lacks the pustules and crusts found in lupoid sycosis and on careful examination presents telangiectasia and follicular plugging as well as typical lesions on the face, ears and elsewhere. Signs of inflammation are much less acute in folliculitis decalvans and its accompanying alopecia is not complete. The tufts of normal hair dividing the cicatricial plaques are absent in lupoid sycosis whose resultant alopecia is always continuous and entire.

Pathology

Various sized plasma and mast cells, and small lymphocytes permeate the epithelial walls of the follicles, rendering them edematous, and at the same time involve the sebaceous and sweat glands. First the pilosebaceous structure and finally the elastic and connective tissue of the cutis above the level of the bottom of the follicle become involved by the cellular infiltration. After destruction they are replaced by fine scar tissue.

Etiology

It is assumed that unknown microorganisms cause the inflammation which initiates the disease, and since they kill the tissue involved, destroying the follicle and the pilosebaceous structures they are responsible for the resultant permanent alopecia.

Therapy

From a study of the literature, the consensus suggests that the treatment of lupoid sycosis is of little benefit to the patient. Yet, in many instances there is a spontaneous cessation after several years, leaving of course, palm sized plaques of permanent baldness. However, a vigorous approach may be of value in the occasional patient.

As in recurrent furunculosis all measures should be taken to improve the general resistance of the patient. These include adequate clinical and laboratory study to rule out underlying systemic disease or focal infections. The prostate, bladder, and urinary tract should be investigated as well as the more usual sites such as the sinuses, teeth, dental roots, and tonsils. A proper dietary regimen should be instituted and should include an adequate vitamin (especially vitamin A) and mineral content. No clinical benefits have been noted from the use of staphylococcus toxoid and vaccines of various kinds have proved useless.

Local measures should parallel those employed in resistant cases of folliculitis, furunculosis, and sycosis vulgaris. A satisfactory daily routine is the application of an antiseptic wet dressing for thirty minutes every morning and night followed by manual epilation of involved hairs. Satisfactory wet dressings in this disease include

- 1 Alibour water
- Copper sulfate 20
- Zinc sulfate 70
- Camphor water qs 3000
- Dilute 1:10 to 1:20
- 2 Burow's solution diluted 1:5 to 1:10
- 3 Vlemmink's solution (1 tablespoonful to 1 glass of hot water)

Subsequent to the manual epilation the area should be sponged off with the remaining wet dressing solution and an antiseptic ointment applied.

- 1 Quinolol Compound Ointment (25 to 50 per cent) containing 1 to 10 per cent sulfur
 - 2 Antibiotic ointments (bacitracin, terramycin, aureomycin, penicillin)
- In a disease which is so resistant to all forms of treatment a course of

oral or parenteral therapy (intravenous or intramuscular) with antibiotics such as terramycin, aureomycin, and penicillin, or chemotherapeutic agents such as the sulfonamides should also be considered. Even after cure of the disease, a maintenance dose of the effective antibiotic may be necessary for a long period of time in order to prevent recurrences.

- 3 Vioform (3 to 10 per cent) in petrolatum
- 4 Combinations of sulfur (3 to 10 per cent) and resorcin (3 to 5 per cent) in an ointment base
- 5 Various mercurial salves (not with sulfur wet dressings)

These remedies are often sensitizing and irritating and must be employed under careful supervision.

The use of x rays for epilating purposes has been advised in the past. It is a hazardous procedure and fraught with medicolegal complications. It does not cure the disease but increases the incidence of atrophy, scarring, and resistance to subsequent measures. In select instances where the disease is confined to a comparatively small area, fractional doses of x rays (50 to 75 r with an approximate half value layer of 2.0 mm Al) once weekly for four to eight weeks may be of value.

Of merit, in some cases, is the frequent application of carbon dioxide slush to the papulopustular lesions. Light therapy, primarily the use of ultraviolet rays from a source such as the water cooled Kromayer lamp, may also be of value.

5 FOLLICULITIS KELOIDALIS (DERMATITIS PAPILLARIS CAPILLITII, ACNE KELOID)

Clinical Features

This disorder was first described by Kaposi in 1869. He gave it the name of dermatitis papillaris capilliti and this term is useful as a description of the preliminary or actively inflammatory stage. The disease was subsequently described under a different title by Bazin and called acne keloid. This title is also useful if restricted to the burned out or end stages of the disease process. Sutton classified it as folliculitis keloidalis, an excellent term. Hollander lengthened it appropriately to folliculitis keloidalis chronica nuchae. The disease occurs essentially in males and is almost exclusively observed at the nape of the neck along the borders of the hairy scalp.

The first manifestations of the disease are single or grouped follicular papules varying in size from 1 to 3 mm. These lesions multiply, some of them becoming pustular. Their location is invariably along the lines where the collar touches the neck, and as they group and coalesce they form an elevated band which occupies the entire posterior border of the scalp. From the very start, this papular or pustular folliculitis is distinguished by its subacute inflammatory character, its very marked induration and the formation of minute fibrous keloids. These lesions usually shed their central hairs at an early stage although an occasional curled and thickened hair or group of hairs may be present on their surface. The confluence of these lesions results in a horizontal row of smooth hard reddish white papules. In time this elevated band or cushion may extend from one ear to the other with a thickness of 1 to 2.5 cm and a length of 10 to 15 cm. Although cicatricial and

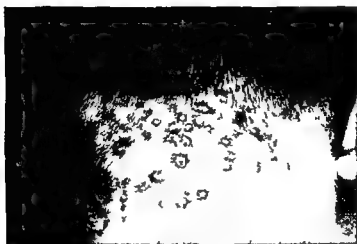


Fig 134—Folliculitis keloidalis Early stage showing many small keloid papules (Courtesy Dr. Marion Sulzberger)



Fig 135—Folliculitis keloidalis Early stage showing single large keloid and satellite inflammatory papulopustules

smooth on its lower aspect this band is a bristly pincushion on its upper side with hairs arranged in tufts or wisps. These hairs not infrequently curl up on the inside and are of surprising length. In time the cushion slowly extends upward over the occipital region never in a downward direction and leaves behind it a pinkish white more or less thickened permanent cicatrix. Surprisingly enough the patients



Fig. 136—Unusual picture of folliculitis keloidalis in a woman following infection due to use of hair clippers. (Courtesy McCarthy, *Diseases of the Hair*.)



Fig. 137—Folliculitis keloidalis. (Courtesy Dr. R. L. Sutton.)

rarely complain of any feeling of discomfort other than that due to the size of the lesions and their need for wider collars. The duration of the affection is indefinite; it may be prolonged for fifteen to twenty years or longer. It is particularly liable to relapse in crops of pustules and papules. Incision of these pustules releases a small amount of pus, but a still larger amount of pus may be discharged by pressure upon the scalp in the vicinity of such lesions. This pus has its origin in local

ized subcutaneous abscesses, probably the focal points for new sites of activity. As the infection subsides, these lesions fill with blood and, in healing, undergo the typical keloidal formation and evolution described above.

Pathology

In the early stage, the lesions show merely a folliculitis or perifolliculitis with an infiltration composed primarily of polymorphonuclear leucocytes. As the disease progresses, the microscopic findings are those of a chronic follicular inflammation noteworthy in that the cellular infiltration is composed almost exclusively of plasma cells, with a few giant cells. Gradually the process leads to the formation of a dense hyperplastic fibrous tissue with masses of plasma cell infiltration and almost total absence of normal dermal structures. The process is different from that of the true keloids.



Fig. 138—Histologic section of folliculitis keloidalis showing extensive and continuous inflammatory reaction in upper portion of cutis. Lower region shows sharply limited cellular infiltrates lying between bands of connective tissue. Note destruction of hair and dilatation of vessels. (Courtesy McCarthy, *Diseases of the Hair*.)

Etiology

The specific microbial agent responsible for this disease has not been demonstrated. It is probably due in part to constant irritation and friction by the collar in association with ingrowing hairs and a low grade bacterial infection. It is fundamentally a disease of young men (third decade especially) with curly, kinky hair.

Therapy

This disease is very resistant to all forms of treatment. Antiseptics, antiparasitics, and chemotherapeutic agents are of value in the very early stages and are

rarely sufficient. The most effective means at this time include manual epilation of the tortuous hairs followed immediately by applications of carbon dioxide slush to the elevated lesions. If this is not effective, radiotherapy should be administered in fractional doses of 75 to 150 r (120 kv, 2 mm Al filt.) for a total dosage of 400 to 600 r. It must be remembered that even such small doses both qualitatively and quantitatively may be followed by permanent alopecia.

In old and long standing cases the best method consists of surgical excision followed by radiotherapy. It is important that the keloids should be removed within the margins of the lesion in order not to stir up further keloidal formation. However, extirpation alone is not to be recommended as it is often followed by recurrence. Radiotherapy should be instituted as soon as a returning elevation of the scars is detected. At this stage the preferred dosage is 150 r (120 kv, 2 mm Al) weekly for a total dose of 600 r. If this course of treatment is adequate only in part because of the age and decreased vascularity of the lesions the remaining keloidal tissue should be treated with local applications of solid carbon dioxide (see therapy of keloids page 504).

Hollander recently reported on the effectiveness of treatment by means of massive electrodesiccation. He destroys as much of the keloidal structure as possible by means of a unipolar electrodesiccating needle and removes the charred tissue with scissors. Subsequent to healing some residual keloid usually requires a similar removal at long intervals. In view of the fact that this disease occurs almost exclusively in young men he also gave his patients oral estrogens (10 mg of diethyl stilbestrol twice daily for an eight month period).

■ DISSECTING CELLULITIS OF THE SCALP (PERIFOLLICULITIS CAPITIS ABSCEDENS FT SUFFODIENS)

Clinical Features

In 1921 Wise and Parkhurst described under the title *A Rare Form of Suppurating and Cicatrizing Disease of the Scalp* a process involving the vertex and characterized by numerous nodules ranging in size from 0.5 to 5.0 cm. Some of these nodules were solid and firm while others were fluctuant. Pressure exerted on a soft lesion resulted in the expression of a seropurulent discharge from sinus openings either at the base of the nodule or at a distance of from 2 to 7.5 cm indicating that the nodules had coalesced in the deeper layers of the scalp. Some of the sinuses could be probed for from 1 to 7.5 cm with relatively little pain. The probing demonstrated a discharge which was occasionally thick and slimy, often mixed with blood and originating in the fluctuant nodular swellings. In time these nodules often become confluent to form elevated sausage shaped ridges with intervening angry looking valleys of red crusted and thickened scalp. In old infections numerous patches of cicatricial alopecia are also scattered throughout the scalp. The scalp margins and adjoining skin often show pustular and impetiginous lesions. A cervical adenitis is not uncommon. The surfaces of the nodules are hairless while in the depressions between them the hair remains but can be removed by slight traction.

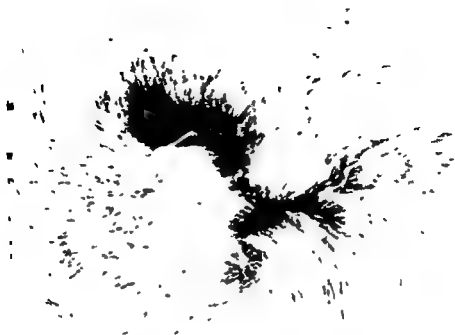


Fig 139 —*Perifolliculitis capitis* (dissecting cellulitis) Early stage showing pus filled burrow with surrounding patch of cicatricial alopecia (Courtesy Dr Frank Combes)



Fig 140 —*Perifolliculitis capitis* (dissecting cellulitis) Ulceration, abscess formation, and cicatricial alopecia in an acute phase of the disease (Courtesy Dr Frank Combes)



Fig 141 —Perifolliculitis capitis (dissecting cellulitis) Advanced stage with numerous pus filled canals, keloids, abscess formation, and cicatricial alopecia (Courtesy Clay-Adams Co)



Fig 142 —Perifolliculitis capitis (dissecting cellulitis) showing firm, elevated ridges and nodules (Courtesy McCarthy Diseases of the Hair Original photograph by Dr H Barney)

The disease is found only in males usually between the ages of 20 and 40 years. The process is essentially chronic in its course, lasting months or years, and recurrences are common. After healing has taken place, there remain various sized atrophic scars together with firm elevated nodules and ridges. This unusual appearance of the scalp during the clinical course of the disease and after involution has taken place has been compared by many observers to the convolutions of the brain.



Fig. 143—Perifolliculitis capitis (dissecting cellulitis). Advanced stage showing cicatricial alopecia and extreme degree of keloid formation. (Courtesy Dr. V. Boghossian.)

The condition might be confused with multiple furunculosis and cyst formation of the scalp, but in the latter there is an absence of the dissecting and undermining elements and an absence of keloid formation during the active stages of the infection while pain is usually a prominent symptom. Moreover, in multiple furunculosis the hair between the furuncles remains healthy and does not loosen in the follicles. In perifolliculitis nearly all of the hairs in the affected area of the scalp may be pulled out with slight traction or may fall out in the course of the disease.

The clinical similarity between perifolliculitis capitis and dermatitis papillaris capitis has been repeatedly emphasized. The variations in the clinical appearance of these processes seem definite enough to differentiate them. Dermatitis papillaris capitis begins as papules or pustules the size of a pinhead usually involving the nuchal region. Later these lesions coalesce, forming keloidlike papilloma

tous elevations studded with firmly embedded hair clusters. Perifolliculitis capitis involves chiefly the occiput or vertex where it occurs as large fluctuant abscesses with deep undermining of the scalp. The hair in the involved area is loosely embedded. The tendency to connective tissue hypertrophy is characteristic of dermatitis papillaris capitis and is less so in perifolliculitis capitis. The differences seen clinically are also apparent histologically. The end stages of perifolliculitis capitis lead to cicatricial alopecia but there should be no confusion between this disease and processes such as pseudopelade and folliculitis decalvans.

The chronicity of the disease is indicated by the clinical picture. Even with adequate treatment the disease lasts for months sometimes years. The difficulty experienced in emptying out the more deeply infected areas and completely eradicating the infection accounts for the marked tendency to recurrences so often noticed. Fortunately the infection, even in severe cases, tends to remain localized to the scalp and the prognosis as to the final outcome is always favorable. Locally a great deal of destruction of tissue with scarring and complete loss of hair may always be expected, as in any other perifolliculitis of the scalp.

Pathology

This disease begins as a folliculitis with disintegration of the follicular wall. This is followed by a severe perifolliculitis with purulent destruction of the deep part of the follicle and disintegration of the hair. The process then spreads into the deep layers of the scalp as a cellulitis producing the undermining seen clinically. Histologically, the chronic process is that of a granuloma. The chronic type of infiltrate observed in the deeper layers of every section is characterized chiefly by small lymphocytes, plasma cells and giant cells and is in part due to the disintegration of hairs which act as foreign bodies. The disintegration of hair appears in the chronic infiltrate of every section either as small fragments or as clumps of brown pigment. The inflammatory process leads to a deep seated fibrosis.

Etiology

Various organisms predominantly streptococci and staphylococci have been cultivated from these lesions. They are responsible for the initial folliculitis and subsequent deeper infection. The dissecting feature of the cellulitis is due to the density of the upper portion of the scalp because of the close approximation and massive distribution of the hairs and their follicles. Such an anatomic structure makes it easier for an infection to spread out and downward.

The entire process may be a severe form of conglobate acne localized to the scalp. If so the androgen-estrogen balance may be causally related. Another etiologic suggestion links the process to hidradenitis suppurativa by presuming the condition to be an infection of deeply situated apocrine glands occasionally encountered in the scalp.

Therapy

This disease is notoriously resistant to all forms of therapy. In selective cases various combinations of chemotherapeutic drugs and local applications may be of

fective. The pyogenic element responds locally to hot wet dressings and irrigations of tyrothricin solution, aqueous bacitracin and penicillin, Thiersch's solution, alibour water, and dilute Vlemminckx's solution. These applications should be made at frequent intervals and syringed through the draining sinuses and pustulonodular lesions. In one instance, I observed rapid improvement following the use of wet dressings and irrigations composed of a 15 per cent sodium propionate solution. In addition, it is essential to institute a complete course of one of the more potent chemotherapeutic agents (i.e., penicillin, aureomycin, terramycin, streptomycin). Specific immunologic procedures are rarely of value unless a pure culture of one organism is isolated from several of the lesions and an autogenous vaccine can be prepared. This procedure should be used as an accessory agent and should merely supplement, never supplant, appropriate antibiotic measures.

7 ANTHRAX (MALIGNANT PUSTULE)

Clinical Features

This disease commonly affects the exposed parts, especially the face and hands. The scalp is an uncommon site but it has been reported. The disease may be considered as an acute infectious disorder, often fatal, which begins as a rapidly necrosing carbuncle with swelling and infection of the neighboring lymph glands. At its onset, it resembles an exaggerated insect bite. However, this impression is dispelled by its subsequent development into an indurated pustule with a dark brown or purple center. Within a short period of time, the central pustule ruptures and is followed by the presence of a dark brown eschar. As the lesion spreads, it develops a central slough encircled by vesicles. The affected portion of the scalp becomes extremely tender and pruritic, and the hair falls out. At this time the margins of the lesion are the site of a dark red inflammatory edema often with lymphangitis and extreme swelling of the neighboring glands. The slough is finally shed and the localized gangrenous process increases in depth and extent. The patient at this time displays signs of serious illness such as high fever, weak, irregular pulse, prostration, the mortality is high. The duration of this illness varies from twenty-four hours in fulminating cases to twelve or fifteen days in the milder forms.

Pathology

According to Unna, the process is a serofibrinous inflammation which rapidly leads to necrosis of the invaded tissue. The corium and hypodermis show marked vascular dilatation and interstitial edema, with enormous numbers of bacilli in the subpapillary vascular region.

Etiology

Anthrax is due to the *Bacillus anthracis*. In man, this inoculation almost invariably results from the occupational handling of diseased animals, such as sheep, goats and horses with splenic fever, or especially of their hides, where the very resistant anthrax spores persist indefinitely. It has also been reported following infection from contaminated shaving brushes. It is most often encountered in butchers, tanners, veterinarians, and those in occupations associated with wool sorting.

Therapy

In former years the only means of treatment consisted of excision or cauterization of the entire lesion. However, the modern treatment evolves around the use of the appropriate chemotherapeutic agent. A considerable number of cures have already been reported following the use of adequate dosages of penicillin, streptomycin, terramycin, and aureomycin.

II ERYSIPELAS

The glazed and elevated red border of erysipelas terminates sharply at the hairline of the scalp. In exceptional instances, this does not occur and the victim is in for a turbulent session. The scalp as well as the entire head, balloons the facial appearance into a grotesque, puffed caricature of itself with enormously swollen ears, lips, and tightly shut, edematous eyelids. During convalescence, there occurs a diffuse but temporary loss of hair. Erysipelas of the scalp may also occur as a localized process following infection of, or injury to, the scalp.

The disease responds promptly to antibiotic therapy as described in standard dermatologic texts.

9 INFECTIOUS ECZEMATOID DERMATITIS

This condition has been described under a variety of names, some of which include chronic streptococcal dermatitis, impetiginized dermatitis, and eczematous pyodermitis. It is a form of pyogenic skin infection, characterized by the appearance of spreading erythema, serous exudation, vesicles, vesicopustules, and yellow crusts. It may appear on the scalp secondary to a pyogenic infection of the ear canal or, not infrequently, as a complication of pediculosis capitis, seborrheic dermatitis or almost any pruritic scalp disorder. The eruption is fundamentally due to secondary infection from staphylococci or streptococci. It is ushered in by local discomfort in the form of itching, burning and tenseness of the scalp and often of the ears. Involvement of the latter is a not uncommon cause of external otitis. Fever, headache and local induration are also frequently noted. When fully developed on the scalp the patches are crusted, weeping or scaling and they enlarge at their margins by the formation of new pustules or vesicles or by an extending erythema as in erysipelas. As the lesions spread, the surface is covered by a sticky, seropurulent discharge which oozes from various points and forms adherent crusts around the hair follicles. Impetiginization often becomes such a prominent feature of the scalp eruption that it masks the diagnosis of the underlying disease. The therapy of this eruption is similar to that described under the heading of Bacterial Infections of the scalp (pages 248 and 255). In many instances wet dressings of a weak sodium propionate solution (1 to 5 per cent) or silver nitrate solution (0.1 to 0.5 per cent) in combination with 1 to 3 per cent Vioform or Dioquin in petrolatum may clear up a long standing and resistant eruption. Small doses of x-ray (50 r once weekly for two to three weeks) may also be of value. When the involvement of the scalp affects the ear as well, antibiotic drops or ear wicks saturated with the appropriate antibiotic agent, depending on cultural find

ings) are efficacious if used in conjunction with the other measures previously described. Following relief of the infectious eczematoid dermatitis, treatment must be directed toward cure or control of the underlying primary disease.



Fig. 144—Infectious eczematoid dermatitis of the face and scalp secondary to a furuncular (staphylococcal) infection of the chin. (Courtesy Sutton and Sutton. *Ill. Cases of the Skin*.)

10 TUBERCULOSIS

Tuberculosis of the skin and especially of the scalp is not very common in this country and for this reason it is usually not well differentiated into the various types described in the dermatologic texts. An acceptable classification is that proposed by Michelson and Lavmon in which the various types are separated on the basis of prognosis.

- A. Forms that are chronic and progressive, rarely terminating fatally
 - 1. Tuberculosis cutis luposa (lupus vulgaris)
 - 2. Sarcoidosis
- B. Forms which tend to heal
 - 1. Relatively rapidly
 - a. Primary cutaneous tuberculous complex
 - b. Tuberculosis cutis verrucosa
 - c. Tuberculosis cutis lichenoides (lichen scrofulosorum)
 - d. Tuberculosis cutis papulonecrotica (necrotic papular tuberculid)

2 More slowly

a Tuberculosis colliquativa (scrofuloderma)

b Erythema induratum

c Tuberculosis miliaris disseminata faciei (lupoid papular tuberculid)

C Forms which usually terminate fatally

1 Acute military tuberculosis

2 Tuberculosis cutis orificialis

a **Lupus Vulgaris**—This slowly progressive disease is characterized by erythematous indurated plaques composed of soft 'apple jelly' nodules. The face is the most common site. Scarring and distortion of diseased tissues often lead to characteristic deformities such as a beaklike nose and ectropion of the eyelids. Involvement of the scalp is not common unless spread has occurred from a patch of lupus involving the lobule of the ear, a favorite site for this disease. At a later period, either with or without ulceration, the auricle may disappear or be reduced to a shrunken shell, the external auditory meatus being occluded by the same process. The scalp as well as the cheek and neck may be invaded by a gradual, slow extension of this process from the ear and appear as a scaly, atrophic, cicatricial area of alopecia in whose margins may be found active reddish brown nodules. The alopecia is permanent. It is very unusual to encounter an isolated patch of lupus vulgaris involving the scalp alone.

b **Sarcoidosis**—Although this disease may occur in association with tuberculosis, leprosy, and other diseases, it cannot be considered due to one specific agent. Sarcoidosis sometimes involves the scalp by extension from a lesion on the forehead. The skin lesions appear as papules, nodules, or infiltrated plaques of an irregular oval shape and a reddish purple color. They are usually present on the arms, shoulders, or face and typically do not ulcerate although they are often followed by atrophy and a white scar. The scalp involvement is of a marginal type on the basis of direct extension from a lesion on the forehead. In some instances, one or several psoriasisform plaques may be observed in the scalp. The scalp involvement is usually associated with alopecia directly over the involved area and is eventually the site of a small patch of cicatricial alopecia.

c **Tuberculosis Cutis Verrucosa**—This form of inoculation tuberculosis is manifested by a hyperkeratotic, warty, pigmented lesion, often fissured and occasionally inflamed, pustular, and crusted. The appearance of this lesion on the scalp is extremely rare.

d **Tuberculosis Cutis Papulonecrotica**—In one form of this type of tuberculosis, usually referred to as acneitis, the lesions consist of firm, brownish red papules scattered over the face. On healing, they leave a small pigmented scar. It is not unusual to find a few of these papules in the scalp margins.

e **Tuberculosis Colliquativa (Scrofuloderma)**—This disease is characterized by involvement of the skin due to direct extension from tuberculous lymph nodes or bones. The enlarged and doughy nodes communicate with the skin through a



Fig 145 —Tuberculosis cutis papulonecrotica (acnitis) with typical, firm necrotic centered papules and minute scars (Courtesy Dr Frank Combes)



Fig 146 —Tuberculosis colliquativa (scrofuloderma) with extension into the scalp (Courtesy Dr Frank Combes)

series of ulcers and reddish granular sinuses which eventuate in rough cordlike scars. The posterior and lateral margins of the scalp are occasionally involved by the extension of this disease and its accompanying subcutaneous nodular gummas, ulcers and fistulous tracts from the cervical auricular, and postoccipital glands.

f Tuberculosis Miliaris Disseminata Faciei—This is a hematogenous form of tuberculosis manifested by crops of small reddish brown maculopapules involving the face. They may appear suddenly in association with an exacerbation of pulmonary tuberculosis and may extend into the scalp.

g Rosacea like Tuberculid (Lewandowsky)—This uncommon disorder is characterized by a facial eruption composed of minute brownish red papulopustules. They occur in large numbers and the surrounding skin is often hyperemic. A few lesions may also be present along the borders of the scalp.

11 LEPROSY

Clinical Features

This disease is now classified under the heading of lepromatous tuberculoid and indeterminate variants of the infection.

According to Canizares the International Congress on Leprosy described the characteristics of these classes of leprosy as follows:

Lepromatous type: Minimal resistance to multiplication and dissemination of bacilli; constant presence of large numbers of bacilli in lesions with distinctive tendency to form globi; characteristic clinical manifestations in the skin, mucosa and peripheral nerves; and involvement of other organs; regular failure to react with lepromin; pathognomonic granulomatous structure of the lesions; definite stability of type and tendency to progression; infectious.

Tuberculoid type: High resistance to growth and spread of bacilli, usually few or no bacilli in lesions; characteristic clinical manifestations mainly in skin and nerves but of limited extent and varying degree; high percentage of lepromin reactions; tuberculoid granulomatous structure in active lesions; marked stability and strong tendency to regression; usually noninfectious.

Indeterminate group: Variable resistance; clinical manifestations chiefly in skin and nerves; skin lesions predominantly flat macules; usually few or no bacilli in lesions; lepromin reaction usually negative or slightly positive; simple inflammatory nature; histologically uncertain prognosis; usually noninfectious.

The predominantly lepromatous forms are characterized by an early macular eruption with eventual infiltration by reddish brown, oily, or varnished nodules. These lesions are not limited to the sites of the previous macules, and their area of predilection is the face. The resultant facial deformity (leonine facies) is severe and grotesque, with nodules studding the nose and ears. The eyelashes and outer third of the eyebrows frequently fall out, a phenomenon referred to as madarosis. A few nodules are uncommonly encountered in the scalp where they appear as pea-sized or larger, soft, smooth elevations not covered by hair. Rarely, large infiltrated plaques of alopecia are present and may go on to ulceration and scar formation.

If the ulceration is absorbed centrally, an annular disc surrounding a cicatricial scar is the sequel.

The neural manifestations of leprosy include symptoms of irritation and degeneration of peripheral nerves such as anesthesia, muscular atrophy, paralysis and mutilation by loss of parts. During the course of predominantly neural forms of the disease, a brownish macular eruption with light centers and dark margins is frequently seen. The lesions are often anesthetic. It is of interest to note that in the anesthetic area the hair turns white and may be absent or brittle and the sweat glands cease to function. The scalp is rarely involved by this type of lesion but may develop a diffuse alopecia following the course of its arterial blood supply.



Fig. 147

Fig. 147—Leprosy. Lesions usually involve the face and ears with avoidance of the scalp. (Courtesy Dr. Frank Combes.)



Fig. 148

Fig. 148—Leprosy illustrating occasional involvement of scalp by cutaneous lesions. This type of case simulates jaw appearance.

Pathology

In the lepromatous forms lepra bacilli are found in considerable numbers in all diseased tissue and especially in cutaneous nodules and nasal ulcers. In the nodules there is a diffuse cellular infiltration of the upper half of the corium composed chiefly of epithelioid and connective tissue cells and large clear lepra cells of Virchow. The latter cells contain numerous compact groups of lepra bacilli and degenerated masses of organism known as clots of Neisser. The nuclei of the lepra cells may undergo hyaline degeneration to form Russell bodies. The in-

filtrate is usually present in distinct microscopic nodules somewhat resembling the naked tubercles of sarcoid. Unlike sarcoid tubercles, the connective tissue criss crosses the cellular mass and the epithelioid cells (actually histiocytes) undergo a peculiar fatty degeneration or infiltration giving them a vacuolated appearance. In addition special stains reveal the Hansen bacilli. The tuberculoid types show few bacilli and a granulomatous, tuberculoid structure.

Etiology

Leprosy is produced by a bacillus (*Mycobacterium leprae*) discovered by Hansen in 1874.

Therapy

Modern treatment of leprosy evolves around general hygienic measures, adequate diet including the administration of vitamins, calcium and iron, and the elimination of intercurrent infections. Specific measures consist of the administration of various sulfones such as Sulphethrone in the form of a 50 per cent aqueous solution and a 25 per cent emulsion for parenteral administration. The dosage is usually 5 to 10 cc twice weekly for several years. Diasone is another effective sulfone and is given orally as 0.10 Gm tablets three times daily for three weeks, then no medication the fourth week followed by a repetition of the schedule. The average total dose over an eighteen month period is 320 Gm. Promacetin, a new sulfone offers even greater therapeutic promise, as does thiacetazone. The latter drug has two marked advantages over the sulfones in that it does not cause mental depression nor is there any evidence that it causes anemia or lepra fever.

II SPIROCHETAL INFECTIONS

1 SYPHILIS

Clinical Features

From the earliest times the loss of hair in victims of syphilis has been considered a noteworthy symptom of the disease. Fracastorius, one of the earliest syphilographers in fact the first to call venereal disease syphilis wrote as follows: "The sick begin to shed their hair not only of their heads but of their eyebrows and beards by which they are made ridiculous in the world some appearing with bald pates although young men others without eyebrows and others again with smooth chins like women or boys." At the present stage of our knowledge we realize that Fracastorius confused many other diseases notably alopecia areata with syphilis and we attach comparatively little diagnostic import to specific alopecia. However, it is important to recognize that the hair and scalp are sometimes involved in a characteristic fashion during the course of the various stages of syphilis. This involvement is usually transitory and leaves no permanent sequelae unless complicated by changes of an infectious or cicatricial nature.

In primary syphilis the scalp may be the very unusual site of an extragenital chancre. The lesion may vary from a mere superficial abrasion to an indurated deep ulcer of the Hunterian type. The regional glands (postauricular or occipital



Fig 149—The moth-eaten alopecia of the secondary stage of syphilis (Courtesy Dr A. Boghosian)

depending on the area involved) are considerably enlarged hard painless and freely movable. If the chancre is ulcerative the subsequent site is usually cicatricial and persists as a patch of alopecia with atrophy of the affected skin.

The diagnosis is made by dark field examination for *Treponema pallidum* and should be performed on any suspiciously indolent lesion of the scalp that shows no tendency to heal. The differential diagnosis is further simplified by the history and occurrence of the lesion within a few weeks following exposure such as a human bite the painless and noninflammatory nature of the lesion the thickening or induration of its base and the enlarged adjacent lymphatic glands. The chief cause of trouble is confusion with an epithelioma and the latter must always be considered in diagnosis and if necessary ruled out by the performance of a biopsy examination.



Fig. 150 — Moth eaten alopecia in a 7 year-old boy. The primary lesion was present on the patient's back. (Courtesy Dr. W. J. O'Donovan. The Har. J. & A. Church. London. Case of Dr. Sequerra.)

Secondary syphilis often gives rise to scalp and hair changes. Involvement of the bones of the skull may cause a dull remittent headache during the early period of secondary syphilis. The hair itself is usually lusterless, dry, and lifeless. Even if it does not fall out spontaneously it is so loosely held in the follicles that it comes out easily if pulled. Alopecia is not uncommon and is exhibited in two separate ways. The usual type is merely a diffuse and general thinning of the hair which occurs during the third to sixth month of the disease. The eyebrows, face, and pubes may share in this generalized alopecia and although unusual an alopecia totalis or universalis may ensue. In this type of alopecia an associated seborrheic disorder is not uncommon and local treatment for that disease should be vigorously

pursued during this complication of syphilis. The reason for this suggestion is that although the syphilitic alopecia disappears with therapy, in the occasional patient the hair may permanently remain more or less scanty. Less often but very characteristically, syphilitic alopecia appears as ragged ill defined, and incomplete patches especially at the back and borders of the scalp as if it had spread up from the nape of the neck. The coalescence of these patches gives the scalp a moth eaten



Fig 151 — Moth-eaten alopecia and elementary syphilitic of secondary syphilis (Courtesy Dr F Reiss)

or mangy appearance. Although these plaques of alopecia may resemble the larger patches seen in alopecia areata it is usually of value to remember that in syphilis we merely see a thinning of the hair and not a complete baldness in any area also in syphilis the patches are more numerous less circumscribed less polished in appearance show no exclamation point hairs and there is no tendency of the hair to regrow without pigmentation. If we consider the pathology of the disease an

interpretation of the nature of this type of alopecia is easily understood. A preceding macular lesion in the scalp can easily pass unrecognized in the hairy regions. However, it upsets the normal nutritive and metabolic balance of the follicle and papilla, resulting in loss of luster of the hair as well as in brittleness and hair fall. These changes affect only the preceding macular lesions, thus the result is a scattering of well separated patches of partial baldness. The reason for the predilection of these moth eaten patches for the back of the head and scalp borders, as if it had spread upward from the nape of the neck, is that the latter is a favorite site for a roseolar eruption. While advanced as another cause of this type of syphilitic alopecia, syphilitic involvement of the autonomic nervous system without any actual local syphilitic process in the hair follicle or scalp. The alopecia appears to occur



Fig. 157.—Frambes form syphilide of the scalp. (Courtesy Dr. W. J. O'Donovan. *The Hair*.)

far more often in association with syphilis of the central nervous system and examination of the cerebrospinal fluid is always indicated in alopecia of this type despite negative serologic findings. This type of alopecia may sometimes be associated with a pigmentary syphiloderm of the neck, as evidenced by either a well marked broad band of pigmentation extending around the neck (collar of Venus) or leucodermic patches surrounded by a network of brown pigmentation.

Alopecia of a cicatricial nature also occurs as a late manifestation (second to fifth year) following healing of pustular and ulcero-nodular lesions. The atrophic pigmented scars left on the scalp and body are indelible and characteristic. The cicatrix and areola are dark brown in the center and become lighter at the margins of the scar. The surface is thin, dull white and slightly scaly, somewhat resembling a coin sized sheet of mica. The late impetiginous syphilide appears in the scalp as

a discrete pustule with a tenacious greenish crust. This heavy crust falls off in time to leave a copper red spot and, eventually, a small white patch of cicatricial alopecia, if the inflammation or ulceration has been sufficiently severe.

Mention should also be made of the so called *corona venerea*. This venereal crown is a massed aggregation of syphilitic papules spread along the forehead at the scalp margins in an irregular band or crownlike arrangement. Where the lesions extend into the scalp as well they are present as scaly, maculopapular groups of seborrheiform appearance.



Fig 453—Annular lesions of the face and scalp in secondary syphilis. (Courtesy Dr V. Boghosian.)

Tertiary syphilis of the scalp is characterized by the gumma. In its early stages this lesion insidiously appears as one of several painless, indolent nodules on the scalp or elsewhere on the body surface. Slowly, but surely, these nodules increase in size and the hair overlying the elevation gradually falls out. The tumor continues to grow and may even attain the proportions of an egg, roughly resembling a sebaceous cyst. Although at first unattached to the scalp, these lesions later fuse with it and change its color to an angry red or purple. Finally this expanding mass bursts through several openings on the surface and discharges the thick gummy secretion from which its name is derived. The resulting ulcers often fall



Fig 154 —Gumma of the forehead and scalp (Courtesy Dr V. Boghosian)



Fig 155 · Serpiginous ulcerative syphilis of the face and scalp



Fig 156—Syphilitic destruction of the face with involvement of the scalp and loss of hair (Courtesy Sutton and Sutton *Diseases of the Skin* Original photograph by Dr J Perkins)



Fig 157—Alopecia totalis due to congenital syphilis. Brother and sister similarly affected (Courtesy Dr W J O'Donovan *The Hair*)



A



B

Fig 158 — Alopecia due to congenital syphilis A Before treatment B After treatment
(Courtesy Dr W J O'Donovan The Har)

away from the surface down through several fistulous tracts with clean cut or punched out margins to the bony skull itself. The intervening skin is often undermined and usually melts down into the ulcerous tracts to form a large necrotic mass. When healing finally occurs (spontaneously or therapeutically induced) the ulcer closes in to form a centrally light brown cicatrix with paler margins made up of white atrophic slightly puckered skin completely devoid of hair. The scars of tuberculous ulcers hold a purplish red tinge for years in contrast to the ultimately white syphilitic scar. Occasionally the scar is considerably depressed and may be attached to the periosteum of the skull. In the unusual case, a diffuse gummatous infiltration may involve large portions of the face and scalp with the formation of large groups of ulcers and extensive loss of tissue. These gummatous patches usually assume the configuration of parts or confluent segments of a circle. The end result of such extensive gummatous infiltration may be almost complete destruction of the scalp and face. It should be mentioned that the diagnosis of gumma is not always clarified by the performance of serologic examinations inasmuch as a certain percentage of these syphilitic lesions occur in individuals with negative serologic findings and therefore must be interpreted as only one point of evidence in the elucidation of a given case.



Fig. 159—Alopecia due to congenital syphilis of the thyroid gland. (Courtesy Dr. W. J. O'Donovan, The Har.)

Congenital syphilis is similar to acquired syphilis as far as the appearance of the manifestations on the scalp are concerned. Of course no primary lesions manifest themselves and the cutaneous lesions appear with little or no respect for proper chronological order but the fundamental morphologic features remain the same. The hair is often affected in syphilitic infants. Although it is particularly liable to fall from the sides and vertex of the scalp it may also affect not only the entire

scalp but the eyebrows as well. Thinning and partial loss of the eyebrows in an infant a few months old is always suggestive of syphilis. On the other hand an unusually abundant crown of dark hair at birth is well known to occur in congenital syphilis it is sometimes referred to as the syphilitic mop or wig and is often subsequently followed by alopecia. It rarely leads to complete baldness but the hair may be left so sparse as to attract attention. Syphilitic bullae with secondary infection on the scalp may leave small cicatricial patches of alopecia. In the occasional case the scalp is also involved in the early and extensive cutaneous syphilides of infantile syphilis. The scalp exhibits thick tough scales which are less adherent and less waxy than in eczema or seborrheic dermatitis and induration if present leads to alopecia.



Fig. 160—Gummatous osteitis and alopecia due to congenital syphilis. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Dr. G. Wende.)

Infantile syphilis is also responsible for cranial deformities affecting the appearance of the head and scalp. These include hypertrophic osteitis of the frontal and parietal bones (olymphant brow) and moderate cranial expansion secondary to the hydrocephalus following syphilitic meningitis. The hydrocephalic head is diffusely thinned of hair and blood vessels show through the thin stretched scalp.

Witkop—This disorder resembles favus and occurs extensively in British Bechuanaland exclusively among syphilitic natives. They refer to it as witkop dikwakwadi or white head.

In a typical case the entire scalp is covered with a thick dry dirty white crust whose surface is irregularly elevated or depressed resembling a relief map. Numerous, small craters may be present and at times the crust becomes stony hard. The superficial layers are easily scraped off but the deeper portions are tough and firmly adherent to the scalp. Where the crust has been removed the underlying scalp shows a complete alopecia with patchy erythema. Some hair remains at the scalp margins and around the ears in a bandlike fashion.

There are no subjective symptoms. The disorder always occurs in syphilitics, usually of the congenital type between the ages of 2 and 10 years, and appears to be noninfectious. A fungus of questionable nature has been isolated from some cases but has not proved to have any causal relationship to the disease. Patients always have a positive Wassermann reaction and often present other signs of syphilis. The disorder responds to arsphenamine and probably would fare equally well on penicillin and other antibiotics as yet not employed in this disease.



Fig 161—Witkop. A Active stage resembling perifolliculitis capitis (dissecting cellulitis). B Healed stage showing cicatricial alopecia. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Dr J. A. Mitchell, Pretoria.)

Pathology

When the alopecia is not associated with an actual syphilitic lesion the scalp shows no abnormal changes. The hair merely shows a type of change observed with many types of toxic agents (exanthems, high fevers, etc.). In other words, the papilla atrophies and disappears as does the hair.

When the changes in the scalp are associated with actual syphilitic lesions, the process is that of the stage of syphilis. In primary syphilis, the epidermis undergoes thinning and erosion. The corium presents a massive infiltration composed of many small round cells, wandering connective tissue cells, plasma cells, polymorphonuclear leucocytes, eosinophils, and new blood vessels. Outside of the cellular mass the pre-existing blood vessels develop thickened walls with beading of the intimal nuclei. Around these vessels there is a collarette or mantle of plasma cells.

In secondary syphilis the scalp lesions are not granulomatous but show an exudative inflammatory process similar to erythema multiforme. The changes are

primarily in the corium where the blood vessels are surrounded by a plasma cell infiltration. Their walls are thickened and exhibit a proliferative tendency.

In tertiary syphilis, the epidermic changes are secondary to those of the corium. The latter show patchy areas of granuloma composed of new blood vessels, plasma cells, small round cells and wandering connective tissue cells. The pre-existing vessels show thickening with an obliterative endarteritis and perivascular infiltrate of plasma cells. The gumma also exhibits these histologic findings deeper down in the fat septa and extending upward. In addition, epithelioid and occasional giant cells are evident but no true tubercles.

Following ulceration and cicatricial alopecia, the histologic findings are merely scar or secondary granulation tissue with absent hair follicles, sebaceous or sweat glands. This scarred area of permanent baldness is secondary to the ulcerative process and may not occur if treatment is instituted prior to its occurrence.

Etiology

Syphilis is due to the *Treponema pallidum* (*Spirochaeta pallida*).

Therapy

The therapy of syphilitic involvement of the scalp is that of the stage of the disease. At the present time the treatment of syphilis evolves around adequate dosage with penicillin usually in amounts varying from 11 to 12 million units. Arsenic and bismuth are also administered by many syphilographers. Local measures are of no value in syphilitic involvement of the scalp other than to combat seborrheic dermatitis and secondary pyogenic infection. The alopecia which occurs during the secondary stage is evanescent in character and disappears under proper treatment of the infection per se. However, the presence of an associated seborrheic dysfunction requires vigorous local measures directed toward the control of that disorder. The cicatricial lesions following ulceration and secondary infection are permanent in nature and their extent depends on the tissue destroyed. Therapy is ineffectual.

2 YAWS (FRAMBESIA)

This specific infectious disease of the tropics is due to a spiral parasite, *Treponema pertenue*, closely resembling the causative agent of syphilis. The disease is largely confined to the Negro races and is common in children. It is transmitted by contact and the initial lesion (mother yaw) is a conical papule usually situated on the trunk and according to Castellani always extra-entail. It may appear also about the mouth or the breast. The lesions may be single or multiple and subsequently become moist, eventuating in a crusted yellowish surface surmounting an ulcerous base. The secondary eruption appears several weeks or months after the primary lesion and is ushered in by joint and head pains and moderate fever. The eruption is usually generalized though the face, extremities and mucocutaneous junctions are the most common sites. The lesions are crusted, granulomatous papules with reddish, fungating surfaces and are not uncommonly observed on the scalp.

They may become confluent to form raspberry or cauliflower like masses. Although the Wassermann reaction is often positive the disease may be distinguished by the extragenital mode of infection in yaws the keratotic lesions of the soles and the absence of mucosal lesions or adenopathy. The tertiary stage resembles syphilis in the development of osteitis dactylitis ulcerated gummas and juxta articular nodules (subcutaneous freely movable firm fibroid tumors near joints). Both the gummas and juxta articular nodules may occur on the scalp. Unlike syphilis yaws is never a congenital disease involvement of the central nervous system is uncommon and alopecia is rarely encountered.

III VIRUS INFECTIONS

1 DISSEMINATED CUTANEOUS HERPES SIMPLEX (KAPOSIS VARICELLIFORM ERUPTION)

If this virus disease occurs in an individual with preceding eczematous patches involving the scalp the latter will also be involved by the characteristic varicelliform or varioliform eruption.



Fig. 167.—Disseminated cutaneous herpes simplex (Kaposi's varicelliform eruption) involving the face and scalp. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Dr. W. H. Brown.)

2. HERPES ZOSTER AND SIMPLEX

The neurotropic virus causing this disease occasionally infects nerves supplying the scalp. *Zoster capillitis* indicates involvement of the second branch of the fifth cranial nerve with lesions on the anterior and posterior scalp, *zoster frontalis* affects the supraorbital nerve (first branch of the seventh cranial nerve) with lesions from the upper eyelid to the vertex in a triangle including half of the brow, forehead, and scalp, *zoster ophthalmicus* involves the ophthalmic division of the fifth nerve and affects the eye, eyelid, eyebrow, and forehead extending into the scalp

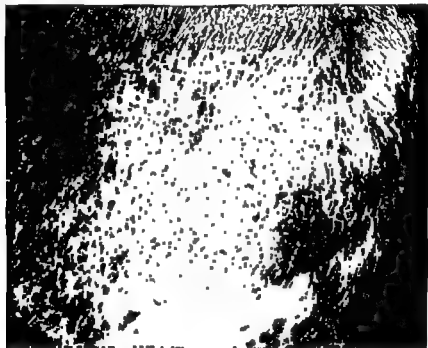


Fig 163—Disseminated cutaneous herpes simplex involving forehead with superficially ulcerated lesions in scalp

The lesions consist of grouped vesicles on an erythematous base, but occasionally a more severe inflammatory or even gangrenous reaction ensues with subsequent disfiguring scars and small patches of cicatricial alopecia

The scalp may also be the site of a few aberrant varicelliform vesicles of the type sometimes encountered in patients with herpes zoster affecting another part of the body

Herpes simplex rarely affects the scalp

3. VARICELLA AND VARIOLA

The scalp often shares in the involvement of the rest of the body by these exanthems. The lesions are similar to those present on the glabrous skin and may be erythematous, vesicular, pustular, or crusted. Healing is often followed by the

appearance of minute patches of cicatricial alopecia at the sites of preceding lesions. Defluvium may also occur during the course of the disease or during the period of convalescence.

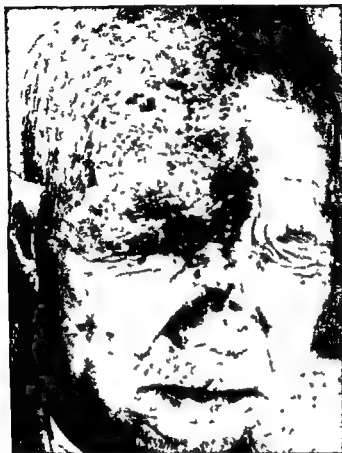


Fig. 164—Herpes zoster involving face and scalp. (Courtesy Dr. Frank Combes.)

IV. PARASITIC INFECTIONS AND INFESTATIONS

1. VEGETABLE PARASITES (FUNGI)

Tinea capitis is a superficial fungus infection of the scalp which principally involves the hair and occurs in children before the age of puberty. It is usually characterized by the friability of the infected hair, its loss in patches, and a moderate degree of inflammation expressed in scalp erythematous lesions. Occasionally, depending on the causative fungus, the inflammation may be acute, with an attendant exudative and secondarily infected eruption. Any area of the scalp may become the site of tinea capitis, although on first examination the patches are most often located on the occipital or temporal regions or on the nape of the neck. The disease may spread to the eyebrows, eyelashes, and even the glabrous skin where circinate scaling lesions may appear.

MICROSPORUM AND TRICHOPHYTON

Clinical Features

There are various types of tinea capitis, depending on the *Microsporum* or *Trichophyton* responsible, each of which presents individual clinical features. The most common, "gray patch" ringworm, is seldom discovered at onset. By the time it is noticed, there may be one or several patches where the hairs have become so dry and brittle that they have broken off a millimeter or so above the scalp's surface. In a few days these patches become circular in shape and are covered with gray scales through which protrude the broken stumps of the infected hair shafts. Occasionally these infected hairs attain a length of 1 cm., and even more. Areas such as these may spread peripherally, coalesce, and, in severe cases, involve the entire scalp. On the other hand, the patches may be accompanied by single infected hairs or groups of hairs situated in regions which are otherwise normal. These are dull and lusterless compared with the healthy surrounding hairs.

This type of "gray patch" ringworm, transmitted by human beings, is caused by the *Microsporum audouinii*. It seldom exhibits any but the mildest inflammatory reactions on the scalp, and pustulation is usually absent. Kerion formation due to this organism has been reported but is extremely uncommon.

The second type of ringworm most commonly encountered is that which is transmitted not only from child to child but by domestic animals such as dogs or cats, and its causative factor is almost always *Microsporum lanosum* or *canis*. It differs from simple "gray patch" tinea largely in the greater degree of inflammatory response which it arouses. The hair stubs in the center of the lesions may be entirely absent. This results in a complete central alopecia due to the falling out of the infected hairs because of the perifollicular inflammation. In such cases the borders, sometimes raised above the central denuded area, contain the broken off infected hairs. The lesion is often covered with scales or even crusts, and minute vesicles and pustules may be observed in this area as well as in the erythematous periphery of the patch.

This form of ringworm often involves the adjacent or distant glabrous skin and manifests its greater propensity for inflammatory response there as well as on the scalp. Kerion is present in only one out of thirty cases. It is not unusual for a contagious factor to manifest itself, with the glabrous skin of the mother and small siblings becoming infected.

A much more serious clinical picture is presented at times by unusually virulent fungi of both human or animal origin. Primary lesions are immediately acute and, although more than one area of the scalp may be affected, these more severe manifestations generally remain localized. The infecting microorganism, which may be either of the *Microsporum* or *Trichophyton* type, sets up an acute tissue reaction which presents itself as a boggy, elevated erythematous, localized tumefaction studded with broken or unbroken hairs and deeply seated vesicles and pustules. Scaling and crusting are both present in exaggerated forms, causing intense discomfort followed by self-inflicted trauma which often results in secondary infections. This acute type of tissue reaction is reminiscent of the granulomatous tinea



Fig 165—Tinea capitis due to *M. audouinii*



Fig 166—Tinea capitis due to *M. lanosum* (Courtesy Clay Adams Co)

profunda of Majocchi on the glabrous skin. Celsus first described these boggy indurated areas on the scalp and they are now known as kerion celsi.

A fourth clinical picture is presented by the entity referred to as black dot ringworm for which one of the endothrix *Trichophyta* is usually responsible. The endothrix character of the invading fungus either causes the hair to break off level with the skin surface or just below it or causes the hair to double back on itself. The tiny rough points which dot the skin resemble comedones or keratosis pilaris, but close observation reveals their true nature. Many long healthy hairs may survive in the same region either singly or grouped and the accompanying scaldiness may be considered dandruff so that the infection often progresses without treatment for many years. When this happens the end result may be atrophy of the long infected hair follicles with scar tissue and minute patches of permanent alopecia. While the other three clinical types are seen in patients before the age of puberty this variety like favus may continue beyond puberty but unlike favus never results in the formation of scutula.



Fig 167—Kerion due to *M. lanosum*. (Courtesy Dr E. Mandell)

Differential Diagnosis

Whenever the examination of a child reveals discrete scaling lesions of the scalp involving loss of hair the first diagnostic possibility is tinea capitis. When the patches show no sign of inflammation they might suggest alopecia areata except that alopecia areata appears suddenly, the involved areas are covered with normal skin and do not have the evidence of broken hairs or a scaly surface. The margins of the patch of alopecia areata usually reveal typical exclamation point hairs. Trichotillomania and trichokryptomania both present broken hairs but no scaling and the hairs involved are normally on the anterior portions of the scalp within

easy reach of the patient's right hand. *Trichorrhexis nodosa* with its broken off hairs is easily distinguished by the fraying of the broken ends and the easily discovered nodules of the hair shaft. *Monilethrix* at times produces a degree of alopecia but the nodules alternating with the tenuous constrictions of the shaft present a string of beads effect which is unmistakable. When a patchy loss of hair is present with inflammatory symptoms it might be confused with pyoderma. However pyodermic lesions on a child's scalp are usually due to pediculosis and the eggs are obvious upon close inspection.

After the clinical features have been determined and fungus infection seems to be the causative factor exact diagnosis must be made with three invaluable diagnostic procedures: (1) Wood's light which is a filtered ultraviolet radiation detecting the presence and extent of the infection; (2) the direct microscopic examination of the suspected hairs in the laboratory; and (3) growth of the organism on suitable culture media.

The ultraviolet light activates a fluorescent element in the hair shaft produced by the fungus infection. Felsher found that the green fluorescent compound from *Microsporum* infected hair belongs to the group of so called fluorescent indicator substances which are easily extracted by hot water or cold 2N sodium bromide solution. This fluorescent material may be a secretion or excretion product of fungus metabolism but Felsher is not yet certain whether the hair proteins are involved in its production. He further discovered that the color of the fluorescence changed with the pH of the solution: a water solution was green at pH 5.5 and blue at pH 8.0. The ions CN^- , SCN^- and I^- suppress fluorescence while Br^- and Cl^- fail to do so. Fluorescence was easily quenched *in vivo* in one day by 10 to 20 per cent potassium iodide in fungicidal ointment massaged into the scalp. When washed out the glow returned within twelve to twenty four hours. It is therefore important to understand that ointments containing iodide may conceal the true nature of the fungus infection under the Wood light and their use should be avoided before testing. On the other hand pomades and tonics containing salts of quinine, petrolatum or mineral oil will give a blue fluorescence which might lead to a serious diagnostic error among the inexperienced. In any event the hair should be thoroughly cleansed before submitting it to the Wood light.

Costello and Luttenberger, working with cases of tinea capitis at Bellevue Hospital, employed a Wood filter which transmitted the bands around 3660 Å. They made the point that fluorescent effects under Wood's light depend on the differing angstrom unit transmission and color comparisons may be made only when similar strengths are being employed. When either organic or inorganic matter absorbs ultraviolet rays of fixed wave lengths and converts them into longer wave lengths this conversion of energy is called fluorescence. Colors and shades of the remitted light vary, depending on the chemistry of the composition and many substances have their own characteristic glow. Visible light rays hinder perfect fluorescence so a dark glass filter made of sodium barium silicate with 11 per cent nickel oxide is placed before the ultraviolet bulb. The filter is practically opaque to the visible light generated in the bulb but transmits the bands at 3600 Å the most effective wave lengths for producing fluorescence.

Wood's light when applied to normal hair, makes it appear dull and lusterless. White hair fluoresces brilliantly, but since the detection of fungus infections is chiefly confined to children before puberty, this is not a confusing situation. Under the light, hairs infected by *M. lanosum* and *M. audouinii* fluoresce with a pale green color. It must be remembered that in the early stages of the infection, the fluorescent color may be noticed only in that part of the infected hair which is nearest the scalp. Wood's light is valuable not only for helping to determine the type of causative fungus but to ascertain the extent of the involvement and, after therapy, to check the completeness of the cure. In cases of *Microsporum* infection with only the slightest clinical evidence, its powers of detection are invaluable. The rapidity of the method recommends it during epidemic situations when large numbers of children must be examined in the briefest amount of time, and individual cultures

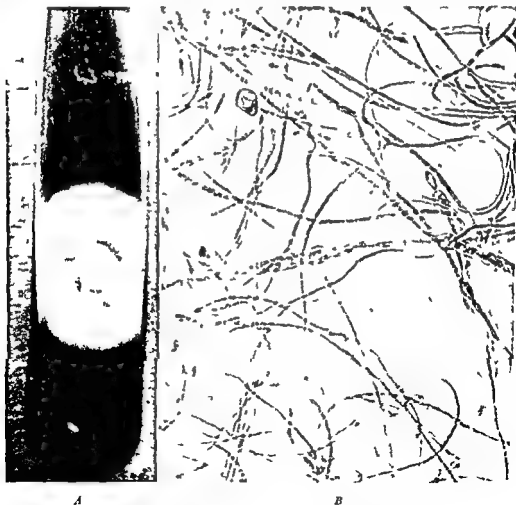


Fig. 168—*Microsporum audouinii*

A Fluffy white culture with radial grooves and concentric rings

B Culture mount with ordinary hyphae, racquet hyphae and chlamydospores

(Courtesy, Dr. F. M. Nelson)



Fig 168, C and D

C, Infected hair showing mosaic sheath of small round spores (similar in both *M. audouinii* and *M. lanosum*)

D, Typical "gray patch" tinea capitis due to *M. audouinii*

(C, Courtesy Dr E Muskatblat D from the Clay-Adams collection)

may be out of the question. Not only is it possible to define the extent of the fungus infection on the human scalp but it is also possible to use it on pets when *M. lanosum* is suspected in order to determine if they are responsible for the contagion. It may also be used on inanimate objects suspected of carrying the infected hairs such as combs, brushes, headgear, furniture, and the backs of theater seats.

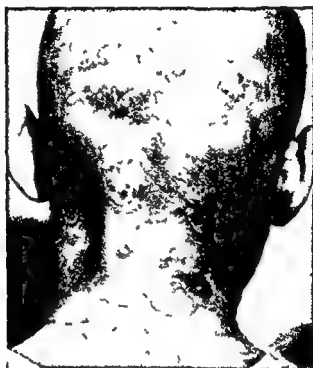


Fig. 169.—Tinea capitis due to *M. audouinii*, unusually extensive case (Courtesy Dr. Miron Sulzberger.)

While Wood's light is entirely reliable with *M. audouinii* and *M. lanosum*, there are fungus infections (including *Microspora* such as *M. fulvum*) which do not fluoresce at all under the filtered ultraviolet rays. If these are diagnosed as a disease other than tinea capitis with no precautions taken against communicability, and the child continues at school, one immediately realizes the danger of such a carrier. Ectothrix *Trichophyton* such as *T. gypsum* and *T. purpurum* are two such offenders. Under the Wood light hairs infected by the fungus appear perfectly normal. *T. cratenforme* fluoresces with a pale grayish light and *T. violaceum* with a dullish white glow or none at all due to the fact that the infected hairs are so deeply imbedded in scales. It is important to use microscopic and cultural procedures on suspected hairs which elude identification.

In the last analysis, subjecting the infected hairs to culture media is the most exact method of revealing the true nature of the microorganism. The time element involved has often discouraged physicians, but since Kligman and Rebell reported on their speeding up process in the routine cultural identification of

Microsporum infections of the scalp, there should be less resistance to this method of revealing the responsible fungus. Identification of *M. audouinii* in the usual manner of planting infected hairs on agar media in a test tube requires seven to fourteen days. Using the same method, *M. lanosum* (*canis*) colonies cannot be recognized until five to eight days have passed. The new method plants the hairs in a drop of liquid medium on a glass slide. The honey media or Sabouraud like media is the most satisfactory.

Honey -----	60	Gm
Neopeptone -----	10	Gm
(Difco)		
Agar -----	15	Gm
Water -----	1	liter



Fig 170—*Microsporum lanosum*

A Luxuriant, downy, fluffy colonies surrounded by yellow pigment

B Typical tapering fuseaux

(Courtesy Dr. E. Muskatblat)

The medium is made up in 250 ml flasks to which streptomycin and penicillin are added in a concentrate of 50 units of each antibiotic per milliliter. The glass slide is mounted on a V shaped piece of glass tubing in a Petri dish. Two pieces of 19 cm filter paper are placed on the bottom of the dish and the dish and its contents are sterilized.

Just before using enough sterile water to wet the filter paper liberally is poured from a flask into the Petri dish which thus serves as a moist chamber. Two moder-

A



B

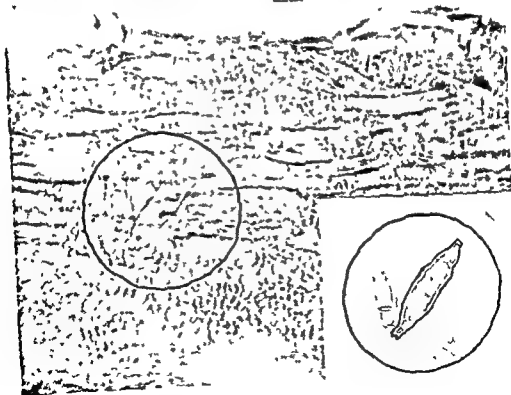
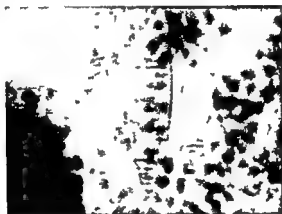


Fig 171, A and B (For legend see opposite page)

ately large drops of culture medium are placed 1 cm apart on the slide with a sterile pipette. The agar in the medium prevents the drops from spreading over the slide. One or two infected hairs are placed in each drop and the preparation is incubated at room temperature. The spores of *M lanosum (canis)* regularly germinate in one day. In from two to three days, the brilliant yellow pigment characteristic of this species develops in the medium around the hair shaft. In three to five days the macroconidia are seen abundantly under a low power microscope in the aerial hyphae above the hair. These develop with amazing speed. A three day culture showing no macroconidia may twelve hours later swarm with them.

The slower growing *M audouini* germinates in from one to two days. The coloring has become characteristically cream colored in from five to six. Since macroconidia are not regularly produced in the culture at this time, they cannot be used for identification but, if any question arises they mature on the eighth or ninth day.



C

Fig 171—A C These photographs of material taken from suspected cases of tinea capitis show various modifications of spindle shaped cells along the shafts of the hair. Superficially, they bear some resemblance to the macroconidia produced in culture by *M lanosum* (Courtesy Drs. B. Appel and H. Ansell and Arch. Dermat. & Syph.)

It would seem that the more rapid growth is due to the water saturated atmosphere. In order to check their new method, the authors cultured 132 cases of tinea capitis, using the old method as a control. They reported that *M lanosum (canis)* infected hairs may be identified presumptively in two to three days and certainly in three to five days when macroconidia have developed. *M audouini* infected hairs are identified in five to six days by noting the absence of yellow pigment, the lack of macroconidia, the lack of spore forms indicative of a contaminant and the presence of a white or cream color consistent with the appearance of *M audouini* in slide cultures.

Loewenthal also made studies of methods of culture which would shorten the time required to identify the causative microorganism. He observed three freshly isolated strains and three stock colonies of *M audouini* and *M lanosum (canis)* in

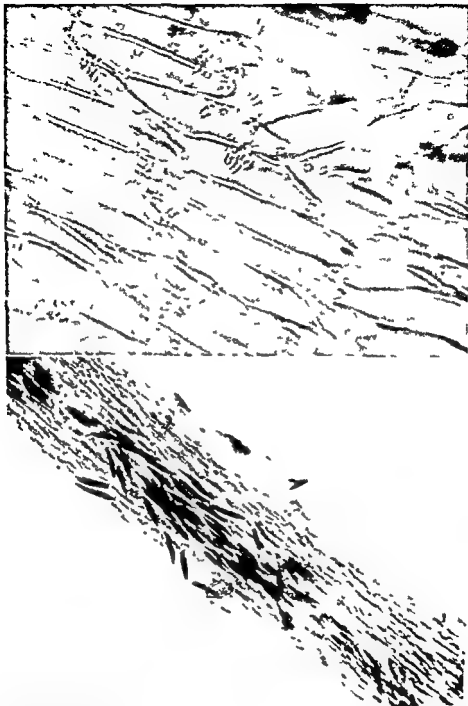
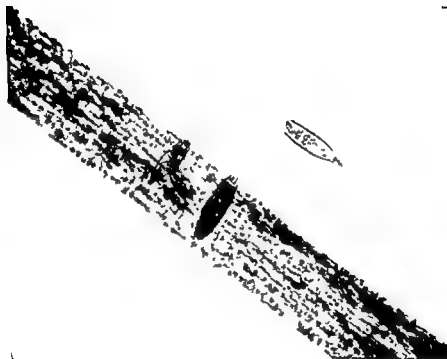


Fig 172 —Higher magnification of spindle shaped cells associated with hair taken from normal and mycotically infected scalps show them as root sheath cuticle cells distinguishable from macroconidia (fusaria) on the basis of comparative size and other criteria (Courtesy Dr L Ajello and J Invest Dermat)

A Group of cells derived from the inner root sheath cuticle of a normal hair

B Hair infected by *M. audouinii* numerous cuticle cells showing size relationship between hair diameter and length of cuticle cells



C

Fig 172—C Macroconidia (fuseaux) of *M. lanosum* taken from an agar culture and placed on a hair to demonstrate the size relationship between hair diameter and macroconidial length. It will be noted that some of the macroconidia exceed in length the diameter of the hair shaft. A comparison of this photograph with the preceding figure points up the size disparity between cuticle cells and macroconidia.

various media, both plain and enriched with yeast extract. It was obvious that the yeast extract both stimulated the growth of the fungus colonies and intensified their pigment production. Contrary to the assumption of Hazen and Benham, the addition of yeast did not abnormally increase the fuseaux which might confuse identification.



Fig. 173.—*Tinea capitis* due to *M. lanosum*. Lesions much more inflammatory in character than in *M. audouinii* as indicated by pustulation, crusting and satellite vesicopustules. (Courtesy McCarthy. *Diseases of the Hair*.)

Since differential diagnosis is most exact for all forms of fungi involved in *tinea capitis* when cultural procedures are employed, it would be intelligent to describe here the typical culture colonies of the species most often responsible. In the excellent volume on mycology by Lewis and Hopper, this subject is discussed in even greater detail but the description is herein limited only to those organisms capable of producing *tinea capitis*. *M. audouinii* in the beginning resembles a white feathery fluff. It grows slowly and becomes grayish white with a moderate degree of aërial growth. There is normally a central elevation with radial grooving, sometimes with secondary radial grooves and concentric rings. Fuseaux and microconidia are either present in only limited numbers or entirely absent. Chlamydospores, racquet mycelium, and pectinate bodies appear especially on a corn meal medium. If allowed to mature for a few days the growth becomes a dull mouse gray throughout, but under Wood's light it fluoresces with bright clear green dots. Cultures do not undergo pleomorphic degeneration.

M. lanosum (*canis*) grows moderately fast in culture (faster than *M. audouinii*). It begins as a luxuriant downy fluff surrounded by yellow pigment and develops an abundant and woolly looking aerial growth, buff tan in color. If allowed to mature for two weeks, the central part of the colony becomes depressed. Radial grooves are normally found, although concentric grooves may also form. The culture mount shows a large number of fuseaux as contrasted with *M. audouinii*. They are tapering in shape with thick, rough walls and from four to twelve compartments. Microconidia, racquet mycelium, and pectinate bodies may also be present. Chlamydospores are also common. When Wood's light is used on the colony, the center of the growth may be lavender blue, shell pink or flesh ochre, the midzone, if there is any, is lavender blue, and the edge an olive drab or mouse gray. Cultures usually become pleomorphic in a few weeks.

M. fulvum (*gypseum*) is one of the easiest fungi to recognize in culture. After two weeks of moderate growth it develops a central limbo, surrounded by a suede like, flat powdery or even granular and discoid growth. Except for the central section which might be white, the characteristic color of the colony is cinnamon brown. It actually resembles coarsely powdered cinnamon. If furrows are present, they are concentric rather than radial. Pleomorphic changes occur easily, as is evidenced by white rifts appearing on the colony's surface. The culture mount shows numerous fuseaux with moderately thick walls and rounded ends. Racquet mycelium, chlamydospores, and nodular organs may be present also, and small round spores in moderate numbers. Wood's light on the colony fluoresces dull but clear with the same cinnamon brown color (*M. fulvum* hairs do not fluoresce). Since nodular organs are not found in other *Microspora* identification is simplified.

*Microsporum ferrugineum** is a causative agent of tinea capitis in China but is extremely uncommon in the Americas. It is culturally typified by a glabrous, nonfluffy colony. The center is brownish red (rusty iron) in color with surrounding cerebriform convolutions of a lighter hue. The slide cultures disclose sterile filaments with large chlamydospores along the sides or at the filamentous ends.

Trichophyton acuminatum is encountered in European cases of tinea capitis. It cannot be differentiated from *T. violaceum* on clinical grounds as they both produce similar types of 'black dot' ringworm. On culture, this endothrix resembles *T. crateriforme* but, unlike the latter, its growth is usually in an upward direction. The color of the colony is usually light or dark ivory with a powdery surface. The center is button or craterlike with a sloping periphery and radial striations.

Trichophyton crateriforme is another cause of ringworm in which the hair stubs are likely to be longer than in *T. violaceum* infections. It grows very slowly and at maturity still covers only a small portion of an agar plate. The central portion of the colony is yellowish and depressed in an abrupt manner, as suggested by its name. The rest of the surface is creamy white and velvety. Small conidia are present either on short stocks or branching from hyphae (thyrses) or in clusters (grappes). Arthrospores and large chlamydospores are common. Under Wood's light the colony appears to be a clear dark olive green throughout.

*Some classify it as but this organism as a *Trichophyton*.

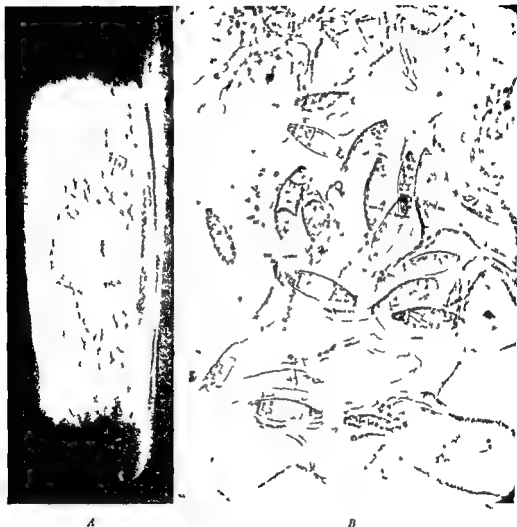


FIG. 174.—*Microsporum fulvum*

A Culture showing flat coarsely powdered (cinnabar) colony

B Culture mount showing numerous fuscaux with rounded ends

(Courtesy Dr. E. Muskatblat)



C

Fig. 174—C Infected hair showing sheath of spores in beaded arrangement (Courtesy Dr. E. Muskatblat)

The growth of *Trichophyton sulfureum* in culture is rather rapid. From a white, fluffy consistency it evolves into a more compact, yellowish gray colony with many radial grooves, central convolution, and surface wrinkling present from the earliest stages. Subcultures may be buff color, less convoluted, and retain their original fluffy surface. Large quantities of pyriform microconidia in open clusters, some fuscaux, and also chlamydospores appear on the culture mount.

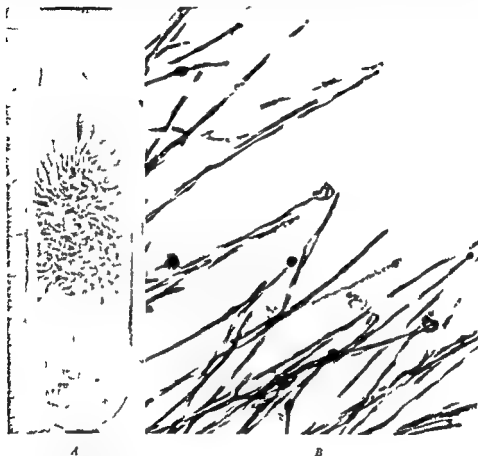


Fig. 175—*Microsporium ferrugineum*. A Culture showing cerebriform convolutions and nonfluffy surface with rusty iron center. B Slide culture with sterile filaments and large chlamydospores along or at end of filaments. (Courtesy Dr. H. Muskatblat.)

Trichophyton violaceum gives rise to a small well defined colony, smooth shiny and a typical deep violet color. Not infrequently the violet color is replaced by a bizarre irregular pigmentation. Convolutions appear in the elevated central area but change to radial grooves near the periphery. Pleomorphism is unusual. The culture mount is distinguished by the absence of microconidia and fuscaux. Numerous irregular mycelia and in older cultures chlamydospores, are present. If the hair is short and twisted, *T. violaceum* is the most likely causative factor.

Trichophyton cerebriforme gives rise to an inflammatory kerionic type of tinea capitis. This neo-endothrix resembles *T. crateriforme* both clinically and culturally.

However, the cerebriform convoluted colonies produced by its cultural growth do not show the depression so characteristic of *T. crateriforme*.

In a recent study by Hligman and Constant they reported that Carrion and Silva have reduced *T. crateriforme*, *T. acuminatum* and *T. cerebriforme* to synonymy under the special term of *T. tonsurans*. The last named species thus resembles *T. mentagrophytes* in that it includes a number of morphologic variants which are not distinctive enough to warrant specific status. It has been shown that colonies kept under cultivation may vary considerably on subculturing so that after a few transfers the original gross characteristics may be lost. An isolate classified originally as *T. acuminatum* may suddenly acquire cultural characteristics which make it more or less identical with strains classified as *T. cerebriforme* or *T. crateriforme*. These so-called species grade imperceptibly into each other. It would seem that too much emphasis has been placed on gross cultural features.



Fig. 176.—Tinea capitis due to *Trichophyton acuminatum*. Note patchy alopecia and small per follicular plugs representing the so-called 'black dots' (Courtesy McCarthy Diseases of the Hair From Sabouraud Les Teignes Masson & Co.)

In Mexico Gonzalez Ochoa and co-workers recently reported 268 cases of tinea capitis 92 per cent of which were due to *T. tonsurans*. Under this species were included among others *T. acuminatum*, *T. crateriforme* and *T. sulfureum* all as variants within the species group. The crateriforme group of Conant and associates which includes four putative species is hence reduced to the single species *T. ton*



Fig 177—*Trichophyton acuminatum* This organism cannot be differentiated from *T. violaceum* except by culture which as shown above has a typical button center and sloping periphery of ivory color (Courtesy Dr F Muskatblat)

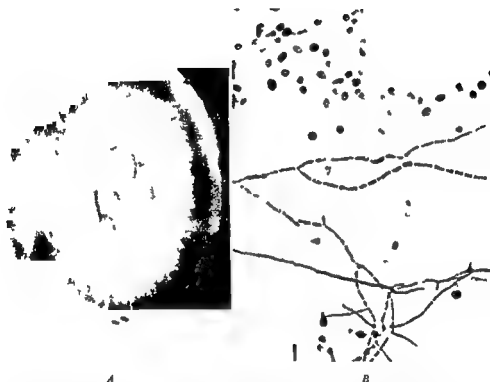


FIG 178—*Trichophyton crateriforme*. A Culture showing craterlike yellowish center and creamy white periphery. B Culture mount with arthrospores and large round chlamydo spores. (Courtesy Dr E. Muskatblat.)

surans Georg one of the outstanding students of the dermatophytes is in accord with this view. Her studies on the nutritional requirements of the dermatophytes have the practical value of indicating means by which characteristic spores may be induced in certain species in which these diagnostic structures are usually rare or absent and thus of leading to clarification of the status and grouping of species for which exact criteria for identification have heretofore not been available.



Fig. 179.—*Tinea capitis* due to *T. sulfureum* (clinically similar to *T. crateriforme* infections)

Kligman and Constant stated that endothrix Trichophyton infections (*T. tonsurans*, *T. violaceum* and *T. schoenleini*) usually show (1) Noninflammatory character of the scalp lesions. Exceptions occur. (2) Negative reactions to trichophyton tests. (3) Difficulty in infecting animals (anthropophilic species). (4) Dull or absent fluorescence. Exceptions have been reported for *T. schoenleini* and *T. violaceum*. (5) Resistance to conservative treatment. (6) Occasional involvement of the adult scalp.

Trichophyton gypsum is rarely found on the scalp where it produces an acute inflammatory kerionic type of tinea capitis. In these scalp infections the growth is of the powdery or granular variety (*T. granulorum*). Culturally it is typified by a cream or light yellow powdery flat and discoid colony. The culture mounts show

trichophytic fuseaux, microconidia as grappes and in series along the filaments (en thyrses). Unlike the other members of the gypseum family, the granular variety shows comparatively few spirals.

Trichophyton faviforme is also a rare cause of acute, inflammatory tinea capitis. The usual sources of this infection are diseased cattle. The cultures show faviforme centers with many convolutions. Their periphery is white and usually surrounded by a third zone of submerged rays. Culture mounts show multiple broomlike branches and swollen, bizarre filaments. Despite all reports to the contrary, typical favic chandeliers may be seen.

Other types of trichophyton, such as *T. purpureum*, affect the scalp with extreme rarity.

Pathology (Hair and Skin)

No matter what species of fungi attack the hairs of man, they originally propagate in the surface epidermis, producing a network of filaments and subsequently penetrating down into the hair follicle. Once in the follicle, they grow deeply and into the hair sheath. From there, they bore through the cuticle and into the shaft itself and then downward toward the bulb, dividing dichotomously. They end abruptly near the bulb, forming a fringelike structure or formation (Adamson's fringe). In spite of the close juxtaposition of this fringe to the hair bulb, it never invades the bulb, so the hair's growth is not interrupted. As the hair grows, it carries the spores of the parasite on to the scalp's surface and beyond, where they have the opportunity of freely infecting healthy hairs and skin.

In the Microspora, the spindle is large, thick walled, and symmetrically fusiform, tapering at the ends, measuring 40 to 120 μ in length by 7 to 30 μ at their greatest width and divided into separate segments by six to fifteen transverse septa. The outer wall exhibits tubercle like prominences. Trichophyta, on the other hand, are thin walled and smooth, shaped more cylindrically or like a distaff. This spore measures about 20 to 50 μ in length by about 4 to 6 μ in width, with two to six transverse septa.

The large, elongated, fusoid, and multicellular spores are not the the only forms characteristic of the whole group of fungi responsible for tinea capitis. Additional structures discernible under the microscope are nodular organs (knots of thickened, interlaced hyphae branches), pectinate bodies (short, thickened, hyphae branches), each in turn producing from its convex side an irregular number of abortive branches of various sizes, resembling the antlers of a stag, and the spiral hyphae, a loose or closely coiled hyphae branch like a miniature bedspring or the tendrils of a clinging plant. Davidson and Gregory demonstrated the adhesive property of the coiled hyphae and suggested that they might account for the means by which a saprophytic growth of fungus in stable refuse clutches at and fixes itself to the hairs of an animal host.

The pathologic findings differ depending on the causative fungus. *M. audouinii* appears as a mosaic sheath around stubby hairs with little tendency to form chains. The spores are round and small (2 to 3 μ) retaining their individual character. Where the glabrous skin is involved mycelium may be detected and even lanugo



Fig 180 *Trichophyton violaceum*

A Culture showing elevated center and smooth concentric rings of a bluish-violet color

B Culture mount showing absence of macroconidia and numerous irregular mycelia, arthrospores and chlamydospores

(Courtesy Dr E Muskatblat)



Fig 180, C and D

C, Infected hairs (*T. acuminatum* is similar) show closely packed spores in hair shaft itself, not on surface of hair. *T. crateriforme* gives a similar appearance on the hair shaft but the spores are more apt to be in a linear arrangement.

D, The typically infected hair responsible for the 'black dot' (Courtesy Dr E. Muskatblat)

hairs are sometimes infected. The sheath formed by *M lanosum* (*canis*) cannot be distinguished from *M audouini*. Mycelium may be noted in sparse amounts in the glabrous skin. *M fulvum* may, on the other hand, present a sheath of spores resembling the beads of a rosary in its earlier development, and also spores with no linear arrangement. After the first stage, it is impossible to distinguish from *M audouini* or *M lanosum* pathologically.

Trichophyton violaceum and *acuminatum* are endothrix trichophyta and thus invade the hair shaft. The spores are larger than those of the microspora (4 to 6 μ) and are arranged in rows or beads or closely packed like nuts in a sack. No fungi whatsoever may be found on the surface of the hairs. In scales or nail tissue, the organism may be present in the form of septate and branched mycelium. *T crateriforme*, also an endothrix like *T violaceum*, reveals large spores in chains or

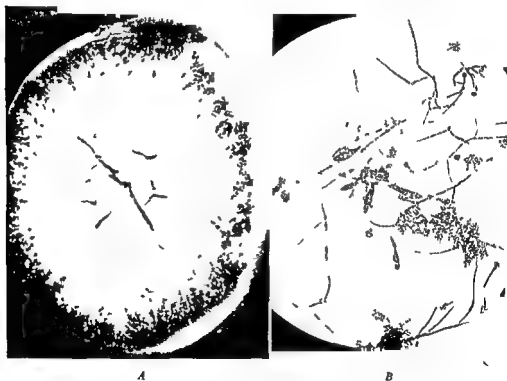


Fig. 181—*Trichophyton gypsum*

A Culture showing powdery granular white colony

B Culture mount showing thin trichophytic fusarux with microconidia (en grappes) and in series on the filament (en thyrses)

(Courtesy Dr. E. Muskatblat)

irregularly in the hair shaft. However, the spores are likely to show a more linear deposition in *T crateriforme* infections and at times the entire hair seems to be filled with fungus elements. *T sulfureum* also gives rise to large spores linearly arranged which invade the hair shaft. When the glabrous skin is involved, filaments are demonstrable in the circinate lesions. *T gypsum* infected hairs show a mass of fungus filaments and spores in chains and clusters outside the hair shaft.

(ectothrix) Air bubbles are common, as in favus, but of a smaller size *T. faviforme* shows a mass of spores, chains, and filaments on and around the infected hair

Microsporum audouinii and the endothrix *Trichophyta*, which are entirely human parasites, create an almost perfect balance between host and parasite, and the infection of the hair seldom elicits an hostile response from the host, which continues to function as such until the advent of adolescence when the reproductive organs' maturation assists its spontaneous cure. While this is a clinical observation which will be discussed more fully under Etiology, it is interesting to note that therapy based on this finding produced no successful results when Lewis and Hopper and others treated young children infected by *M. audouinii* with sex hormones

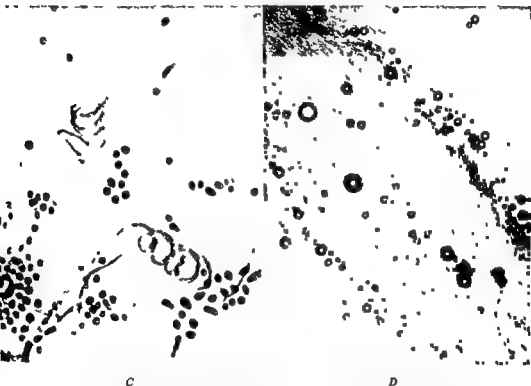
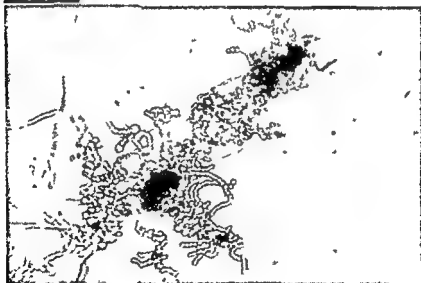
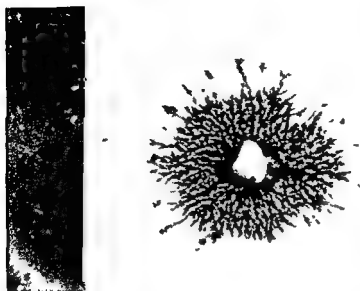


Fig 181—C Culture mount showing characteristic spirals of *T. gypsum*. Unlike other members of the *gypsum* family, the granular variety shows comparatively few spirals. D, Infected hair shows a mass of filaments and spores in chains and clusters outside of the hair shaft. Air bubbles are common as in favus, but are of a smaller size. (Courtesy Dr. H. Muskatblat.)

Etiology

The incidence of the causative fungi varies in different parts of the country and the world. City dwellers suffer from one species while farm laborers suffer from another. In the New York Skin and Cancer Unit 2,857 cases of tinea capitis were studied from 1940 to 1947 and it was found that of these, 83.3 per cent were caused by *M. audouinii*. On the other hand, studies made at the Massachusetts General Hospital showed that *M. lanosum* was responsible for 65.3 per cent of all

A



B

FIG 187—*Trichophyton faviforme* A Culture showing convoluted faviform center and radiating periphery

B C D Culture mounts showing multiple broomlike branches and swollen filaments (Courtesy Dr H G Carney and Arch Dermat & Syph)

C



D



Fig 18? C and D (For legend see opposite page)

cases of tinea capitis in that region. It is also the predominant microorganism in California and other sections of the West and South. Ninety-five per cent of 81 cases of tinea capitis at the Children's Hospital in Havana were due to *M lanosum* and 5 per cent were due to *T gypsum*. *Trichophyton violaceum* as a causative fungus is usually found in Russia, Poland, Italy, or the Near East. In the United States, the organism is chiefly found in immigrants, with sporadic cases reported in native stock.

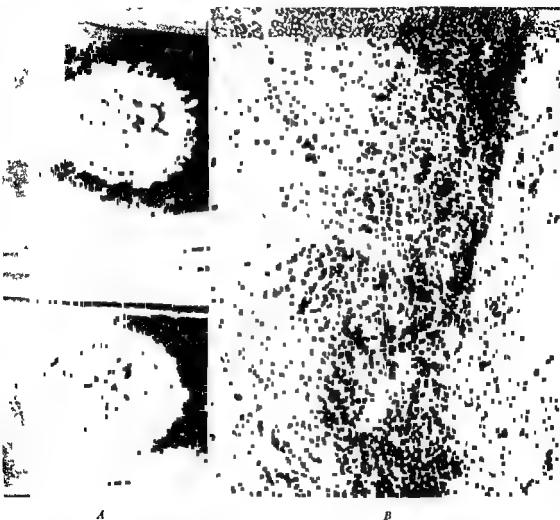


Fig 183—*Trichophyton fauiforme*. A Typical cultures with convoluted centers surrounding white zone and raylike periphery. B Infected hair showing a mass of spores, chains and filaments on and around hair shaft. (Courtesy Dr E. Muskatblat.)

The studies in Boston were with children whose average age was 6 years and 2 months. In this group one and a half times more males were infected than females, although *M lanosum* was equally responsible in both sexes. Other workers reporting cases of *M audouinii* showed that this causative fungus appeared on the scalps of three times as many boys as girls. Crocker's 600 cases indicated a 6 per cent

margin of males over females. Beeson's examples proved to be 85 per cent boys while Pardo-Castello reported a 31:1 ratio on behalf of the boys. Fox and Fowler found the opposite true in the studies of tinea capitis in adult life where 32 out of 48 cases involved women possibly mothers who had taken care of the infected scalps of their children.

Children before puberty are those subject to infection. Unsterilized combs, brushes, and headgear may all be responsible for carrying the fungus from one

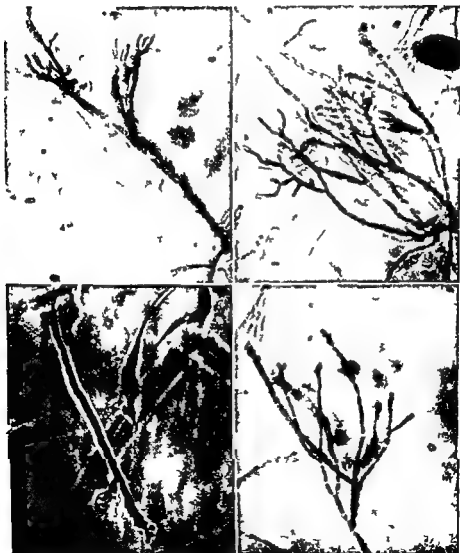


Fig. 184. *Trichophyton fauoi*. These culture mounts prove that this organism definitely produces favic chandeliers despite reports to the contrary. The photographs show multiple broom-like branches with numerous swollen and bizarre filaments. (Courtesy Dr. E. Muskatblat.)

human to another, adding the risk of contagion to groups of children living in close quarters. It is usually only the virulent animal fungi which persist after puberty, when, even without treatment, there may be a spontaneous cure.

M. lanosum (*canis*) readily infects kittens, puppies, and the usual laboratory animals and is therefore known as the animal Microsporum. *M. fulvum* may carry contagion from human beings and animals, but the latter are resistant to infection by *M. audouinii*. *T. crateriforme* may cause lesions in guinea pigs, but they heal quickly and spontaneously. On the other hand, *T. violaceum* has been transferred successfully to guinea pigs, dogs, and cats.

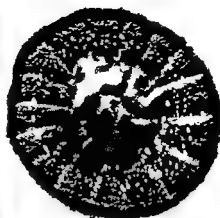


Fig. 185—*Trichophyton cerebriforme* showing convoluted, cerebriform colony. (Courtesy Dr. E. Muskatblat.)

The etiologic question repeatedly asked is: What factors cause the involution and cure of tinea capitis at puberty? The answer lies in the chemical changes present in the scalp upon the maturation of the sex organs and the endocrine glands responsible for their behavior. At the University of Chicago, Rothman studied the fungistatic activity of a series of specific fatty acids ranging from 7 to 13 carbon atoms in molecular structure. He discovered that the odd number fatty acids are the most effective. Comparing the amount of fat contained in the hair of children with that of adults, he discovered that there was approximately five times as much fatty acid in the hair of the latter. He believed that with the stimulation of the apocrine glands attendant upon adolescence, there appeared this increase in the fungistatic substances which cause the final inhibition and elimination of the fungus. Bergeim and Cornblet observed that the human scalp had a distinctly acid reaction.



A



B



C

FIG 186—*Trichophyton purpureum* A Young, fluffy white colony B Older colony with purplish coloration throughout underlying media C Culture mount showing long thin fuscaux (Courtesy Dr E. Muskatblat)

Mycologic methods have recently been adopted by greater numbers of physicians who wish to make more exact diagnoses of the causative fungi in their cases of tinea capitis. Guesswork will no longer satisfy the dermatologist who wishes to enjoy the most effective therapeutic measures and, perhaps, to control the spread of the disease in an epidemic situation. Modern contributions to concepts of mutation and variation of the fungi and their requirements for nourishment are simplifying the classification of these pathogens, which for a long time were treated as individual isolates by a variety of workers.

To recapitulate briefly, the fungi which cause ringworm of the scalp belong to the botanical molds which develop spores and mycelia during their growth and fall into two genera: the *Microspora* and the *Trichophyta*.

The *Microspora*, which include *M. audouinii* and *M. ferrugineum* of human origin, and *M. lanosum* or *canis* and *M. fulvum* (*gypseum*) of animal origin, build a mosaic of spores in a sheath which extends from the mouth of the follicle to the hair bulb. Branching threads of mycelium run longitudinally within the horny part of the hair forming 'Adamson's fringe' just short of the hair bulb.

The *Trichophyta* show either an endothrix or an ectothrix variety. The former consists of spores which invade the hair shaft, but the latter fungus surrounds the shaft, making it less brittle and therefore simpler to epilate. Unlike the endothrix variety, the inflammatory response to the host is acute. Pustulation becomes part of the clinical picture and accounts for the spontaneous expulsion of hairs, which happily curtails the course of the disease. The deep involvement of the scalp sometimes affects the glands of the neck, with resultant lymphadenopathy.

Sabouraud, performing cultures in 500 cases between 1907 and 1909, reported finding forty different species, but, over thirty years later, Macleod and Minende reclassified them in a more concise manner.

Therapy

Although roentgen ray epilation is admittedly the most efficient therapeutic measure in tinea capitis, many articles have been published in the last few years suggesting new topical applications and techniques. So many workers seem to be investigating this field in their desire to avoid the method which requires the most skilled operators, that it is not impossible to envisage a time when a treatment will be found which will perform a cure in as rapid and efficient a manner as the roentgen ray.

Reiss, studying the fungistatic and genestatic effect of steroid hormones on pathogenic fungi, concluded that the use of these hormones not only reduces growth but also becomes responsible for a reduction of the conidial structures which could be interpreted both as evidence of growth stunting and a reduction of potential virulence. There is a marked decrease in the numbers of macroconidia of *M. lanosum* (*canis*) and *M. fulvum* in media containing diethylstilbestrol and estrone. Estrogenic hormones have a similar effect on *M. audouinii*, *T. gypseum*, *T. purpureum*, *T. sulfureum*, *T. crateriforme*, and others. On the other hand, the androgenic genestatic effect is negligible in cases of *M. audouinii*, *M. lanosum* (*canis*), and *T. sulfureum*.

In all inflammatory types of *tinea capitis* and even some of the resistant, scaling variants, local measures should be given a trial for several months. The scalp should be shampooed twice daily and thoroughly anointed with a fungicidal ointment or solution. Too vigorous scrubbing should be avoided. Some of the most effective remedies include salicylanilide (5 per cent), mixtures of the unsaturated fatty acids (propionic, caprylic, undecylenic), iodine (1 to 5 per cent), thymol (1 to 5 per cent), ammoniated mercury (5 to 10 per cent) alone and in combination with salicylic acid (2 to 6 per cent), sulfur (5 to 10 per cent) and chlorhydroxyquinoline ointment. These applications should be performed in conjunction with daily or triweekly observation and manual epilation under Wood's filter. The mother may be instructed in the use of small, flat toothed forceps for removal of fluorescent hairs under this light. The author has seen many localized patches cleaned out by means of careful and diligent measures of this type. The hair must be grasped as far down as possible and removed with gentle traction lest it break, due to the brittle, infected shaft. Resistant hairs may be destroyed by electrolysis or electrodesiccation as described by Costello.

Young recently reported excellent results in a group of recalcitrant *M. audouinii* infections of the scalp. He employed daily applications of 3.3 per cent podophyllo-toxin in 90 per cent ethyl alcohol. The solution must be thoroughly but carefully massaged into the scalp for at least a thirty day period.

There have also been several recent and optimistic reports on the use of Asterol hydrochloride in *tinea capitis*. From personal experience, I believe that these reports require additional confirmation.

Brain, Crow, Haker, McKenny and Hadgraft, seeking a penetrating vehicle which might carry an effective fungicide to the base of the hair follicle and make possible a cure for *tinea capitis* by simpleunction without preliminary epilation, developed such a preparation consisting of 0.5 per cent phenylmercuric nitrate in a base of Carbowax 1500 and Crell #6. The pH is adjusted to between 4 and 5 by adding a solution of citric acid and sodium propionate. The hair was clipped short and kept that way and a linen cap was worn continuously, while each morning the scalp was washed with soap and hot water, and scrubbed with a nail brush. The ointment was applied three times daily with a toothbrush for five minutes. Since pustulation and lesion formation cause natural epilation, inflammation when present was welcomed, and when absent it was provoked. Of eight cases of *M. audouinii*, six were cured in an average of 55 days with no relapses. The two unsuccessful cases were cured eventually by x ray epilation. Of nine cases due to *M. felineum*, eight were cured in an average of 75 days. Of four cases where the fungus could not be identified by culture two were cured by this treatment, and the other two by x ray epilation in four and five months, respectively. A 9 year old girl with *T. endothrix* infection for seven months became clinically and microscopically negative in fifty-three days. The authors concluded that roentgen ray therapy will afford the quickest cure and will continue to do so until more potent fungicides and more penetrating bases might be developed.

Combes and I, in our article entitled 'Technic and Problems of Roentgen Ray Epilation,' considered the aspects of this major therapeutic measure in detail.

PREPARATION OF THE PATIENT

'The preparation of the patient is comparatively simple. The scalp hair should either be shaved and allowed to grow slightly or clipped closely (between 0.3 and 0.6 cm. in length). If the hair is more than (0.3 cm.) in length it acts as a mechanical filter. If there are any signs of inflammation of the scalp, as evidenced by pustulation or thick crusts, it is advisable to wait until these changes have been controlled with wet dressings, antiseptic ointments or other therapeutic measures. If there are large areas of alopecia or definite scars on the scalp, it is always advisable to photograph the patient prior to the epilation in order to avoid medicolegal action in the future. The patient and the parent should always be closely questioned concerning previous treatment and especially about previous roentgen ray epilation. In one instance, a child had been referred from a clinic, where he had been seen by several physicians to the dermatologic radiotherapy department for epilation. The child's head had been shaved and mapped out for the treatment with roentgen rays. The child was so docile and showed so little apprehension that the technician commented on his lack of fear. The child replied that he was not worried because he had had the same treatment three months before. Subsequent investigation proved this to be so. The treatment had been administered at another hospital but the mother was dissatisfied and changed hospitals. By some error, no record of the treatment was forwarded, and but for a chance statement this child might have had permanent alopecia. We routinely question the patients and their parents before the roentgen ray therapy, despite their clinical records. Have you ever had your head shaved? Did you ever lie down under a machine like this? These are routine questions when the patient is brought to the x ray room.

It is still claimed by many authorities that irritants of any type should not be employed before or after the treatment with roentgen rays. However we have advised in hundreds of cases the daily use of tincture of green soap followed by ointments containing [various fungicides such as] 5 and 10 per cent sulfur or ammoniated mercury [and the newer agents] both before and after epilation. There have been no instances of undesirable sequelae from these applications and we believe that our percentage of failures, recurrences and spread to other children has been much smaller because of their usage. For three weeks prior to and following epilation a salve containing 3 per cent ammoniated mercury is advised. For the next three weeks the percentage is increased to 5 and finally to 10. The 10 per cent ammoniated mercury ointment is used until the patient is discharged. [At the present time I prefer mixtures of salicylanilide (5 to 10 per cent) and the various fatty acids caprylic, undecylenic and propionic.]

AGE

The question of the proper age for the performance of epilation with roentgen rays is an important one. Formerly it was stated that if a child approached puberty he should be treated with local measures alone because the infection would disappear spontaneously. This statement is open to question. We have treated a series of patients expectantly but the disease did not respond despite the onset of puberty. In fact we have seen several patients of 14, 15 and 16 years of age and even adults with tinea capitis.

TECHNIC

For children below the age of 3 years or in refractory cases various measures have been suggested. Some therapists prescribe sedatives in an attempt to quiet the patients. We have seen children treated in this manner incapable of following instructions and frequently unable to control their movements. The use of wooden blocks or sandbags are also of little value in controlling the refractory, frightened or extremely young child. In these cases the fields are mapped out and the child

placed in position for the first field after the body has been mummified. The parent is instructed to don a fluoroscopic lead rubber apron and long lead rubber gloves. The child is held without the hands of the operator. This procedure is repeated for the five fields, always screening off the parent's hands with extreme care. The body is protected by means of a lead rubber apron and kept as far from the table as possible. If the assisting parent is female, always ascertain whether she is pregnant, as this would, of course, eliminate her from assisting in this manner. The reason for employing a parent rather than a technician is obvious: the parent is but slightly exposed for merely a single treatment.

The older child is brought into the room and told to sit down on a chair. He is usually apprehensive, and it is of the utmost importance that he be reassured of the fact that the treatment will not hurt. In a small percentage of cases in which the child is nervous and restless it is advisable to do a trial epilation. This is essentially a dress rehearsal of the entire procedure from beginning to end, performing all motions with the exception of actually applying the roentgen rays. Several days later when the child returns for treatment itself he has lost his fear and the performance is repeated with ease. It is also of value to confront the patient with another child of similar age who has already experienced epilation. The other child is questioned in such a manner as to convey the information to the new patient that the treatment is painless. Children will frequently believe one another concerning transactions of this nature, whereas they remain skeptical of the veracity of an adult.

The measurements of the scalp should be carried out as though a game were being played. The child enters readily into the spirit of things, and future procedures are rendered less difficult. It is suggested that the beginning therapist actually measure out the various Kienbock-Adamson points rather than employ one of the time saving devices. It is always useful to be able to employ a technic in the absence of gadgets as the latter may not always be obtainable. In this country the five point method is employed.

The use of thick pastes of bismuth compound or similar substances for marking is not advised inasmuch as they may screen out some of the rays from an underlying infected point. [A wax pencil or gentian violet are good marking agents.] After the operator has become proficient in marking out the fields and knows the method thoroughly it is then permissible for him to employ one of the time saving devices described elsewhere. These are usually obtainable in several sizes and enable one to mark out a scalp in a few minutes. In unusual shaped heads it is always advisable to check the measurements by the original method.

We still prefer the five point technic of Kienbock and Adamson. This method has stood the test of time and is the method of choice for routine scalp epilation. We have employed it personally in the performance of several thousand epilations for ringworm of the scalp and have never seen a patient in whom there was any undesirable permanent effect.

* FORMULA

Various physical formulas have been suggested by different authorities for roentgen ray epilation. [See Chapter III on alopecia due to x rays, page 189.] The various factors will be discussed singly.

The employment of mechanically or thermionically rectified machines makes no difference provided that the calibrations are recent and accurate. It is preferable to employ a machine which operates at a constant potential in order to maintain the voltage across the tube at or near the peak voltage throughout the cycle. This feature has been incorporated in many of the more recently manufactured machines by the proper inclusion of condensers in the rectifying circuit. The proper

kilovoltage to be employed varies with the therapist. We have performed epilations with low voltage (60 kilovolts) and with comparatively high voltage for dermatologic therapy (135 kilovolts). There is no great difference in results as far as we have been able to determine. However routinely we prefer 100 kilovolts. With the low voltage (60), an occasional imperfect epilation may be attributable to the slight degree of penetration of rays generated at this figure. The results at 100 kilovolts show little difference from those at higher figures and the latter have greater potentialities for depth damage because of shorter wavelengths (higher frequency or harder roentgen rays).

The amperage merely expresses the magnitude of the electric current and merits no involved discussion. We usually employ a current of 5 milliamperes.

The time of exposure has always been a subject of discussion. It has been stated that the optimum rate of exposure (exposure per unit time) is 100 r per minute. Accordingly, in the past it was stated that a so called epilation dose (300 r) should be delivered in three minutes. We see no indications for allotting this long period provided that the employment of extreme care and accuracy are not influenced. With refractory children or in active (and careful) clinics time is frequently a valuable quantity. In some instances such as in a young and active child it might mean the difference between the success or failure of an epilation. We have used a dosage rate of 100 r per thirty seconds with success. Of course it is me

the epilation is of course the loosening of the hairs in their follicles. It would seem logical to employ a more penetrating ray by the insertion of filters, in order to achieve the desired quality of radiation. In controlled series the results are comparable regardless of the use of filters. Epilations have been performed with no filtration other than that inherent in the tube and again with 1 to 3 mm of aluminum and even with 0.25 mm of copper and 1 mm of aluminum. The results show little difference. Accordingly we perform our epilations without the use of filters for the reason that potential depth damage is less and the results are just as good or better.

The focus skin distance (distance from focal spot of x ray tube to the skin of the patient) varies with the therapist. We employ a focus skin distance of 20 cm.

The half value layer (thickness of material which reduces to one half the intensity of a particular beam of radiation) must always be stated in order to give information concerning the penetrating power of the beam of radiation. We employ a half value layer of approximately 1 mm of aluminum. Neither the effective wavelength, the effective absorption coefficient nor the half value layer (nor any other method which depends on an average) affords a unique description of the quality of a beam of radiation according to Quimby. A beam generated at high voltage but not filtered so that it has many soft components may have the same average as a beam at lower voltage but filtered so that its softer rays are decreased in importance. For practical purposes however if the voltage is nearly the same beams of the same half value layer are essentially alike in their behavior in tissues. It is good practice to specify voltage filter and half value layer.

To recapitulate the other factors include 100 kilovolts, 5 milliamperes, no filtration other than that inherent in the tube, 0.5 mm of aluminum, focus skin distance of 20 cm and rate of exposure of 100 r per thirty seconds (350 r in one minute and forty five seconds).

DOSE

It is important to understand first the exact meaning of this term. It is merely the amount of radiation delivered at any particular point. It has been loosely referred to as the amount absorbed by the particular tissue under treatment.

Unfortunately, this is untrue as that particular quantity cannot be measured as yet. We usually refer to the air dose in discussions of treatment, because it can be determined with the greatest accuracy directly from the calibration of the x ray tube. It has little actual relationship to treatment because it is merely the amount of radiation reaching the central point of the skin field. We are actually concerned with the skin dose which is the air dose plus backscatter (radiation scattered back by the tissues). This dose can be measured directly on the patient or calculated from various tables. However, for purposes of discussion we shall adhere to the air dose.

It was formerly taught that 300 r (aerolux) was the dose for the treatment of tinea capitis. Recent experiences in recent epidemics have shown that 350 r is necessary. Various therapists employ 200 to 300 r. We have observed that this amount of radiation gives satisfactory results in both clinic and private practice when the patients are carefully studied and observed following treatment. With this dose, we have not

observed any regrowth of hair. However, this observation obviously contributes little information on the treatment of the entire scalp. We know of and personally observed a single case in which the entire epilation (350 r) was repeated within one week. The error was not discovered until the fifth field was about to be exposed for the second time and this last exposure was not performed. The hair fell out in the customary period of time for a perfect defluvium. Regrowth commenced in three months and was entirely completed by the end of the fourth month. Apparently the margin of safety is much greater than it is generally considered to be although the interval of one week is vastly different from an immediate dose of 700 r. The most intrepid investigator hesitates to experiment with higher doses when the specter of permanent alopecia overshadows his innocent charges. It is sufficient to know that a certain safe dose will be adequate in the majority of cases.

The biologic reaction must also be considered. It has been reported that in persons with fair hair, light complexion and thin skin erythema (from roentgen rays) will develop with a dose of 300 to 350 r. On the other hand in persons with dark hair skin erythema may not develop with less than 500 r. There is a diversity of opinion however, as to whether the threshold erythema of blond and brunet is the same. The dose of 375 r is usually considered safe.

McKee, Matsheller and Capellaro stated that there is a direct relationship between the biologic effect and the number of roentgen units required to obtain such biologic effect. Some ionometers give one reading for a given biologic effect and others give entirely different readings. Commercial ionometers also vary with the mean wavelengths of the x ray beam. Ionometers should be calibrated against an open standard. The basic reaction is the basic reaction for other requires 300 r for the same biologic effect. If the same ionometer were used to calibrate the two x ray machines the number of roentgen units would probably be the same. As an additional safety factor all x ray machines should be calibrated against a biologic standard viz the erythema dose or the epilating dose.

Measurement—After the beginner has mastered the technic of measuring the Kienbock Adamson fields he may employ one of the time saving devices now available. The one we employ consists of three strips of intersecting transparent plastic material joined together to form a small cap.

Fixation of the Head—Various measures have been suggested in order to insure against motion of the patient's head during the course of treatment. Sand bags are frequently employed by being stacked under and along the sides of the head. In our experience they have been a nuisance because of their weight and the blocking of vision. Wooden blocks are used in several institutions and are of some value. We employ a thin but soft pillow.

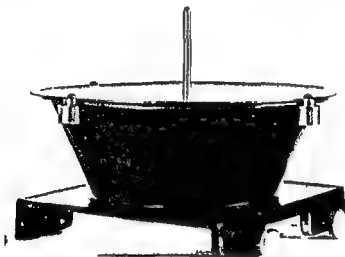


Fig. 190. A device of value in the performance of an x-ray epilation. It consists of a flat metal cone with a pointed rodlike center which is focused on one of the Kienbock Adamson points and slipped off prior to each field exposure.

A certain amount of difficulty is experienced by most operators in directing the x-ray beam perpendicularly to the plane of the particular field under treatment. Some operators merely direct the tube (with either an open port or a large cone) toward the scalp in which

This method is not exact. The tube stand. Whether these

surmount this difficulty first fully employed in the Dermatologic Radiotherapy Clinic of Mount Sinai Hospital. It had been made in response to a request for some method which would lead to more accurate focusing of the roentgen rays on the five points of the Kienbock Adamson fields. Prior to this intersecting silk threads had been placed over the cone in an unsatisfactory effort to locate the center. With the present method a wide cone with a focus skin distance of 15 mm. is placed over the portal of an x-ray tube. The device is so constructed as to slide over this cone. At the edges of this metal sheet are three small metal knobs which enable the device to slide on and off the cone with ease. In the exact center of this metal sheet is a blunt steel rod which extends downward for an additional 3 cm. thus increasing the focus skin distance to 20 cm. The roentgenologic technic accordingly incorporates a focus skin distance of 20 cm. among its other physical factors.

In the actual performance of the epilation the child is placed in the proper position and shielded with lead rubber. The device is placed on the cone. The flat surface of the device is then made parallel to the plane of the area of scalp under treatment with the projecting rod touching the respective Kienbock Adamson point. The x ray tube is then locked into position and the device gently removed from the cone. This procedure is repeated for each of the five fields.

POSSIBLE DANGERS OF ROENTGEN RAY EPILATION

Errors might also follow a change from a greater to a lesser distance without modification of the basic formula. It is for this reason that a busy clinic should adopt a single uniform and successful technic for all epilations.

The operator should know that field 3 is followed by 1 then 2 then 4 and finally 5 if this is the sequence adopted by that particular clinic. This detail appears unimportant but may be of critical importance in the midst of an epilation performed by a student or beginner. No epilation should ever be permitted without supervision unless the student has watched ten and performed at least five epilations to the complete satisfaction of the instructor. The formula and setting of the proper dials for the particular technic adopted by the clinic should be checked and rechecked before each treatment. If the machine or machines are in daily use calibrations should be performed at least once a month. The latter insures against a shift or falling off of dosage at the periphery of the area under treatment. Fields should always be measured for each machine in use in order to insure against major variations in dosage at their peripheries.

RESISTANT INFECTIONS FOLLOWING ROENTGEN RAY EPILATION

In a certain percentage of cases the desfluvium may not be complete or reinfection (from contaminated hats, articles of clothing, other children or infected members of the same family) may occur. In the latter cases it cannot be emphasized too frequently that the child who has recently experienced epilation should

every few days or weeks

In those and recheck present they
 children are not advised that the parent may be assisted in the performance of this technic and permitted to work on the child's scalp for fifteen to thirty minutes daily. A sharp pointed splinter forceps is most suitable for this purpose. Small recurrent patches may respond to applications of iodine in an effort to produce a local inflammatory reaction. If several hairs repeatedly show fluorescence it may

be necessary to infiltrate the area with a local anesthetic (2 per cent procaine hydrochloride) and destroy the hairs with electrolysis or electrodesiccation, as suggested by Costello

'The question of further treatment in patients who have already been subjected to a single epilating exposure is a serious one. Opinions differ as to the length of time which should elapse between the first and second exposures. Six months is generally considered to be a safe period, although we prefer nine months to one year. The same techniques and physical factors are employed. We again expose the child to a dosage of 350 r. These patients must be followed with extreme care and thoroughness, inasmuch as a second failure is a serious problem from the viewpoint of physical, mental and public health. Some authorities advise even a third roentgen ray epilation, but we believe the procedure too dangerous



Fig 191—Appearance of the scalp following a properly performed x ray epilation

If the child has run the risk of permanent alopecia on two successive occasions, the performance of a third is suggestive of an invitation to disaster. Fortunately, we have never been faced with this problem. If the necessity arose, we would probably attempt to treat the patient with local measures and manual epilation, regardless of the ultimate prolongation of treatment.

"CRITERIA OF CURE

"In the average case, the patient may be considered cured if an examination under the Wood filter fails to reveal fluorescence of the hair on two successive occasions separated by a three-week interval. This, of course, applies only to the usual types of tinea capitis produced by fungi which exhibit fluorescence under this filter. The other, and fortunately, rare forms of this disease can only be followed by microscopic and cultural examination of the infected hairs. It is important to perform similar examinations, also, in the usual cases of tinea capitis.

"PUBLIC HEALTH ASPECTS

"A group of dermatologists in New York outlined a [series] of measures designed to control subsequent epidemics of tinea capitis. These measures, as advanced by Lewis, Silvers, Capollaro, Muskatblit and Mitchell, are as follows:

"1 *Case Finding*—(a) Tinea capitis should be made a reportable disease. (b) As a preliminary step to control an epidemic of ringworm it is essential to make a city wide case-finding survey of all school children, using filtered ultraviolet rays. (c) The case finding survey should be repeated every three months. (d) The examining team should be equipped and instructed to take material for microscopic examination and cultures when the examination is made. (e) When cases are found, a district diagnostic center should be set up. (f) The city should appropriate sufficient money for these projects. (g) Cultures should be performed before the roentgen ray epulating dose is administered. (h) A pamphlet describing ringworm of the scalp in simple terms should be given to every child in the school before the examining team reaches the school.

"2 *Education of the Public*—The public should be reached through the press, from pamphlets and by the radio. In this way uninfected children and their parents may be apprised of dangerous habits or practices.

"3 *Institutions*—It is recommended that city wide, complete and periodic inspection of children who are in institutions be made by qualified physicians. No child is to return home from an institution unless it has been examined under filtered ultraviolet rays. No child should be accepted for institutional care without a report of freedom from the disease after examination under filtered ultraviolet rays.

"4 *Barber Shops*—(a) The barbers should be told about the epidemic and cautioned to be on the lookout for lesions on the scalp. (b) The barbers should be prohibited from cutting the hair of a child known to have or suspected of having ringworm infection. (c) The possibility of chemical sterilization of barbers' instruments is to be considered.

"5 *Movies and Other Public Places*—Children should wear hats as a precaution against infection in public places, such as picture houses, children's clinics and subways.

"6 *Preparation for Roentgen Ray Treatment*—Clipping and shaving of the hair prior to epilation should be done in the clinics at which infected children are treated, or the parents should be instructed to do so. The parents should be warned not to take an infected child to a barber.

"7 *Treatment*—(a) Roentgen ray epilation is the best available treatment in the majority of cases of ringworm infection of the scalp due to *Microsporum audouinii*. Infection due to *Microsporum lanosum* usually does not require roentgen ray therapy. (b) The Department of Health should register equipped and properly staffed clinics and qualified physicians for the treatment of ringworm of the scalp. It is hoped that the registration itself will act as a deterrent to improper treatment with roentgen rays.

"8 *Suggested Method of Management*—Observation of the patient under filtered ultraviolet rays prior to the administration of roentgen rays is an integral part of the treatment, since it establishes the extent of the infection.

"9 *Criteria for Cure*—It is recommended that no child be permitted to return to school until he is entirely free from infection. At least two examinations with negative results, one week apart, under filtered ultraviolet rays should be made before the child is referred to the

Board of Health for admission to school. A subsequent examination under the filtered ultra violet rays one month after return to school should also be performed.

"Prevention of Tinea Capitis"—In a community in which there are no cases, but in which the children may come into contact with the infection, the school board would be well advised to purchase a Wood light and have the school nurse instructed in its use. The children in the schools could then be periodically examined not less than every three months.

FAVUS

Clinical Features

The type of fungus infection caused by *Achorion (Trichophyton) schoenleinii*, being stubbornly chronic and not self limiting, may persist for twenty or more years. At times it covers much of the scalp, at others it remains localized for as long as five years. Because of this stubborn persistence, a single patient may present a manifold variety of symptoms characterizing all stages of the disease.



Fig 192—Favus. A Showing typical scutula. B Same case following x ray epilation (Courtesy Dr. William Dobes.)

The essential eruption begins at the site of inoculation as a very small erythematous patch which is irregular in outline and slightly elevated. On this circumscribed field there subsequently appear the minute yellow puncta of favus in the horny layer of the epidermis. After two weeks the destructive, almost confluent crusts begin to form about the hair follicles. These crusts range from 1 mm to 1 cm in size, are sulfur yellow in color, and are circular in shape with depressed



Fig 193



Fig 194

Fig 193—Favus in a 6 year old girl. The lesions are somewhat similar clinically to severe seborrheic dermatitis (Courtesy Dr William Dobes)

Fig 194—Long standing untreated favus (Courtesy Dr V Boghosian)



Fig 195—Untreated, self-cured favus of many years duration. The end result is cicatricial alopecia (Courtesy Dr William Dobes)

centers like Lilliputian saucers. The crusts, called scutula, appear to be firmly attached to and imbedded in the epidermis. Each of these concave discs encircles a hair or tuft of several hairs still long (these hairs break off far less frequently than in the *Microsporum* infection), but lusterless and grayish in color. Often dry, gray oases of hair appearing in the midst of normal, shiny hair is the first danger signal recognized by the patient.

When the scutula are removed, and this requires considerable exertion, there remains a corresponding depression sometimes covered with serous or blood stained fluid. The hairs which pierced the scutula usually neither break nor split but they come out with the lining of the follicle as a vitreous sheath. It requires at least several weeks after the onset of the initial eruption for the hairs to show symptoms of infection. The reason for this time lag is due to the fact that the hairs have been at tacked inside the follicle and must grow the entire length of the follicle and through the thickness of the scutula before becoming evident.



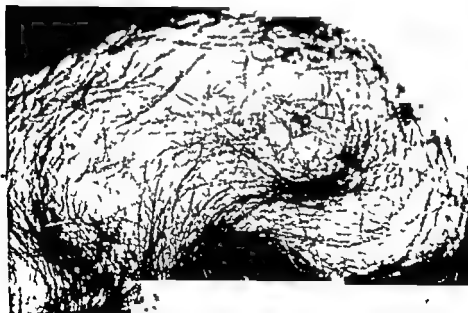
Fig. 196 Favus showing typical scutula. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph by Dr. G. M. MacKee.)

In untreated cases the scutula coalesce, forming thick gray masses composed of fungi and epithelial scales. When these masses are removed they disintegrate into powder under ordinary finger tip pressure. Old cases show small areas of erythema and pustulation surrounding a few infected hairs, alternating with a number of scarred patches of cicatricial alopecia, uneven, ridged and irregular in appearance. The end result is often a scalp with a moth eaten appearance due to these numerous patches of cicatricial alopecia.

Difficulties in diagnosis occur in pityriasisform cases of favus where honey colored scales pick up and hide the minute scutula. Such scales must be removed in order to locate the concave discs at the scalp surface. In order not to confuse



A



B

Fig 197 Fagus showing typical scutula *A* Anterior view *B* Side view (Courtesy Clay Adams Co.)

these cases with long standing seborrheic dermatitis and psoriasis, it is also important to look for infected hairs. All involved hairs are not necessarily infected. During the stages of scar tissue formation, many pale, atrophic hairs are in evidence due to impairment of their nutrition by the slowly cicatrizing scalp.

Further diagnostic difficulties may be met in cases of long duration where the scutula are absent and follicles with dull hairs surrounded by small, inflammatory areas resemble the various pyogenic infections of the scalp. Furthermore, the perifollicular depressions and isolated grayish hairs with erythema about their follicles resemble the scars of acne varioliformis. In all such cases, microscopic and cultural studies are necessary in order to confirm the diagnosis of favus. When the color is indistinct in cases where the scutula are malformed or squamous in character, they may be wiped with alcohol to bring out the original sulfur yellow color.

Early writers make much ado over the 'mousy' odor which they detected emanating from scalps infected by favus. Apparently some cases have it and others do not.

Pathology

The fungus in favus may be found by microscopic examination of the scutula, the infected hair shafts and on the surface of the involved adjacent skin. *Achorion schoenleini* grows within and on the stratum corneum until in contact with a follicular orifice it penetrates the opening and grows down and into the epithelial sheath and the hair shaft itself. The infection inside the hair ends at the neck of the hair bulb in a group of filaments similar to the fringe of Adamson in a Microsporum infection.

The pustules result from the essential inflammatory process when the prickle cell layer of the epithelium becomes edematous and parakeratotic. Between the layers of horn which have been so affected the branched mycelial threads are located. They are composed of cylindrical elements of various size separated by cross walls. Independently situated spores singly in chains or in clusters are also found. The only alteration in the cutis is a slight vascular dilatation and a perivascular infiltration of lymphocytes and leucocytes. As the parasites multiply in the horny layer the edema increases as does the dilatation and infiltration until superficial vesicles are formed (favus herpeticus).

These original vesicles dry out and the disease moves slowly into its next stage known as favus crustosus where the scutula appear as a mixture of mycelial threads, individual spores, leucocytes and degenerated epithelial cells. The pressure of these scutula on the epidermis thins it out to a few remaining rows of atrophied cells. Sometimes the involved areas of stratum mucosum are destroyed so that eventually the scutula rest directly on the supporting tissue of the corium with subsequent vascular changes and infiltration by lymphocytes and plasma cells.

In advanced cases pressure on the papillary bodies of the dermis and the destruction caused by the invading parasite result in almost complete loss of the sebaceous glands and sometimes the elastic tissue. Spontaneous healing is due to the fact that the base of each scutulum is circumscribed by a wall of lymphocytes and leucocytes that successfully keep nourishment from the mycelia and spores causing their demise. Eventually the scutulum is separated from the underlying tis-

sue by a band of cells and falls off. The infiltrate in the cutis then gradually disappears and only the scar tissue remains.

Microscopic examination of the infected hairs after immersion in the potassium hydroxide solution demonstrates the characteristic behavior of the favic invader. Along the entire surface of the hair there are many air bubbles which disappear if they stand too long or if the slide has been overheated. Sabouraud gave the classic explanation for the appearance of these bubbles. In the aerial portion of the hair shafts, there are hollow, slightly wavy linear cavities which are occupied before their death by mycelia and spores, and afterward by air. When the potassium hydroxide is introduced, the air is forced to the surface where it forms the bubbles already noted and which accounts for the grayness of the hair shaft. The fungus in the scutulum shows many closely packed spores and filaments with a radiating corona. When magnified many degrees, the infected hairs appear to be distinctly polymorphous. Eppright and McCuiston, reporting a case of endemic favus in Texas, noted that the Wood filter revealed a dull gray fluorescence of about 60 per cent of the scalp hairs throughout their length. They also found such unusual elasticity in the infected hairs that they compared them to rubber bands.

Some confusion might exist even under the microscope. The mycelial threads divided into chains of spores also occur in endothrix types of *Trichophyton*, but these have no sporulated mycelia anywhere but in the hair itself, and air bubbles are isolated phenomena rather than the most obvious characteristic. In microid *Trichophyton* (*gypseum*) there are masses of fungi outside the hair.

McCarthy has described the growth in culture of *Achorion schoenleini* with great detail. He advises that the parasite be cultured from a section of hair directly above the root at the source of infection so that it may be uncontaminated, since many cases have secondary invaders due to the chronic nature of the disease. A whitish yellow mass, irregular but smooth like the dripping from a wax candle, evolves from a peptone, glucose, and maltose base, covering an area 3 to 5 cm in diameter in three months. Eventually the surface becomes convoluted and powdery and the white darkens to brown. The culture twists into folds radiating outward from the center toward the lower edges and grows down into the media, thereby cracking the agar. When the 4 per cent glucose of Sabouraud's media was replaced by 12 per cent honey, it hastened growth perceptibly. On culture mount, the characteristic favic chandelier may be seen, as well as large numbers of gigantic chlamydospores. Microconidia and fuseaux are seldom seen and rudimentary old cultures may undergo white and fluffy pleomorphic degeneration.

Etiology

The *Achorion schoenleini* was discovered in 1839 by Schoenlein, after whom it was named. If we consider the extrafollicular portion of the hair, the fungus is essentially an endothrix *Trichophyton*. However, the organism is actually an endothrix form if we take into account the masses of fungi in the hair sheaths and epidermis (scutula). This parasitic fungus was first isolated from the blood of patients with favus in 1926 by Ambrosoli and again in 1932 by Louner and Rieff. It has been a disease long associated with poverty, filth, and malnutrition which invariably accompanies the underprivileged. Early cases were usually traceable to the countries of Central Southern and Eastern Europe where miserable conditions

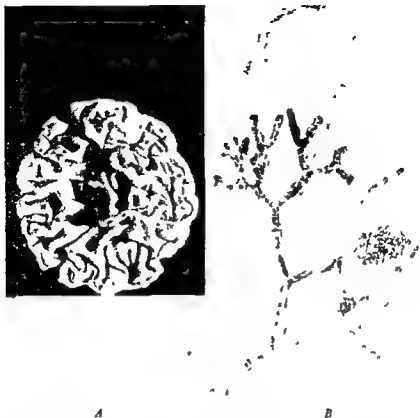
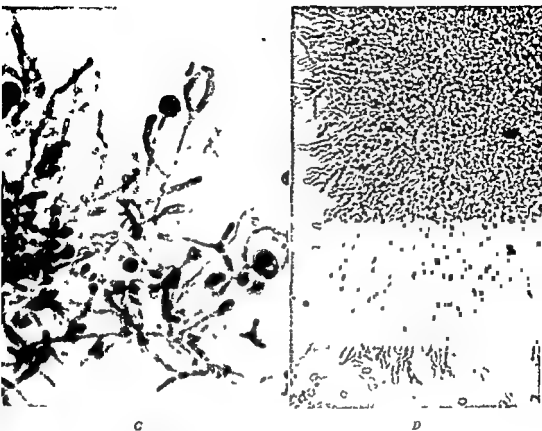


Fig 198—*Favus*: A, Culture showing typical, twisted, convoluted, powdery surfaced colony B Culture mount showing classic favic chandelier C, Culture mount showing gigantic chlamydospores D Margin of scutulum showing many closely packed spores and filaments with a radiating corona (Courtesy Dr E. Muskatblat)

prevailed in overcrowded slums. This is no longer true. Kentucky, California, Georgia and Texas have all had cases in recent times and the ten cases in Georgia in 1918 existed in a family which had been in America for generations with none of the patients ever having been out of the state. For twenty years the Royal Infirmary of Scotland treated 97 cases following an immigration from the Continent in 1918, while there were no cases reported in the three years previous to that date.

Favus spreads by direct contact with infected combs, brushes, and headgear. Although mice, barnyard fowl, dogs and horses suffer from related *Achoria*, it is not the human parasite (*schoenleini*) and therefore transmission from animals is negligible. Some believe that an abrasion of some sort is a prerequisite to infection. In spite of one reported case in a 1 month old baby, *favus* rarely attacks the very young or the very old. When it is found in old people it is usually an avirulent remnant of an earlier active lesion.

Ciarocchi, examining 5574 cases in Rome, decided that a perfectly quiescent focus on the scalp of an aged person may still be the cause of spread among the young. He came to this conclusion because in spite of careful and thorough treatment for affected children and adults, the incidence of *favus* was just as great after ten years of adequate care as it was at the beginning.



C D
Fig 198, C and D (For legend see opposite page)

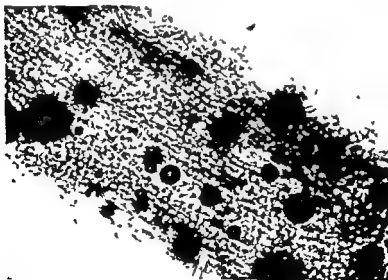


Fig 199—Infected hair (favus) with mycelial threads divided into chains of spores and numerous large air bubbles (Courtesy Dr E Muskatblat)

Therapy

The same therapy is recommended for favus as is used in other resistant cases of tinea capitis, to induce temporary alopecia by roentgen ray epilation and follow this by fungicidal applications until regrowth. Cases with advanced changes will be left with scarred atrichic areas, while improper treatment or neglect eventually result in multiple patches of cicatricial alopecia.

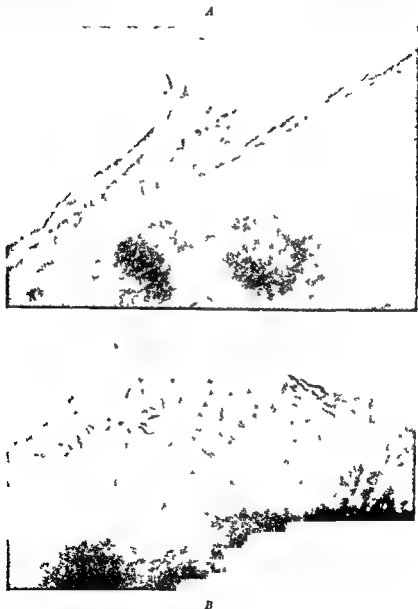


Fig. 200—*Piedra*

A Early stage of invasion of hair shaft (black \square edra)

B, Later stage of invasion of hair shaft (black \square edra)

(Courtesy Dr. E. Muskatblat)

PIEDRA

Clinical Features

Piedra (trichosporosis, *pie*dra nostras, *tinea nodosa*) is a fungus infection limited to the hair shaft itself without involvement of the adjacent scalp. From one to twenty-five stony nodules spaced at intervals of a centimeter appear along the hair shaft. These nodules vary in size from minute, gritty particles imperceptible to the naked eye, to pinhead-sized formations. "Piedra," meaning stone, characterizes the flintlike hardness of the nodules which, no matter how small they may be, are easily felt between the thumb and forefinger and, when hit by a comb being run through the hair, sound like the rattling of pebbles in a bean bag. The nodules, like nits, may adhere to either side of the shaft or form a collar around it.



Fig. 200, C and D

C, Nodule of white piedra on hair shaft (low magnification)

D, Nodule of white piedra on hair shaft (high magnification)

(Courtesy Dr. E. Muskatblat)

Pathology

Under the microscope, in a 10 to 30 per cent solution of potassium hydroxide, one discovers that the hair between the hard discrete nodules is entirely normal. A buff to light green translucent stroma forms an interrupted sheath along the hair shaft. Usually it adheres to the hair under its cuticle layer by means of an agglutinous substance caused by the liquefaction of the cell's external membrane. Less frequently, it may damage the cuticular layer of the shaft at the site of the nodule producing trichorrhexis.

The nodules of *Trichosporum giganteum* are formed of spherical closely packed cells resembling double contoured spores. These cells are round on the outside of the mass and rectangular where they have been compressed within the growth. They measure from 10 to 12 μ in diameter although many larger, multilocular cells are also present. As a result the varying shapes resemble a mosaic pattern. Blastospores occur in the hyphal mat but no asci or ascospores are encountered. Gram positive staphylococci have been found associated with the fungus in some instances and are probably symbiotic invaders of the mucoid material produced by the organism.

The *Trichosporum beigeli* develops rapidly on Sabouraud's medium beginning as a yellowish creamy somewhat moist growth and then as it develops becoming darker dull finely wrinkled and inclined to adhere to the agar.

The nodules of *Piedraia hortai* are microscopically evident as linear masses of rectangular cells with a length of 10 to 12 μ and a diameter of 4 to 7 μ . The mass of cells contains large spherical asci (30 μ in diameter) in which there are eight elongate ascospores approximately 40 μ in length and 6 μ in diameter. These ascospores have long (15 to 20 μ) filiform appendages. According to Moore the whole mass although simulating a sclerotium macroscopically is actually analogous to a perithecial structure. The cultural growth is of a velvety dark brown color with a lighter adherent margin. In time the colony becomes black.

Etiology

The type of piedra which is designated as white is usually encountered in Colombia. It produces grayish or creamy white nodules which are softer than the black variety and usually enveloped with a mucoid substance. Another piedra caused by a different fungus *Piedraia hortai* is known as black and is characterized by hard black and brittle nodules along the hair shaft. The white species has been reported from South America with additional examples from Central Europe, England and even Japan. In 1939 Kneedler found 311 out of 1120 school children in South Siam infested with piedra. It was originally noted in Colombia where it involved the long hair of women who take their shampoos in stagnant river water and then apply a heavy muciluminous grease for huddressing purposes. Kneedler's study indicated that the use of oil was unimportant in the development of the disease in Siam. In Brazil it is seen chiefly in youngsters who use a cactus oil as a hair pomade. In British Guiana the disorder is attributed to a milky white water as it is not encountered among natives using a brown peat bush water. Although the disease is primarily encountered in the tropics it has been observed in Europe and in the temperate zones.



Fig 201—Culture of *T. betgel*: showing creamy finely wrinkled colony (Courtesy Dr E Muskatblat)



Fig 202—Culture of *P. hortai* with velvety dark brown surface (Courtesy Dr E Muskatblat)

The parasite responsible for white piedra belongs to the hyphomycetes group called *Trichosporon giganteum* by Unna in 1895, *Trichosporon granulosum* by Ota in 1928, and *Trichosporon minor* by Leao in 1940. At present, the preferred name is *Trichosporon giganteum*. The member of this group encountered most frequently is the *Trichosporon beigeli*, as it was termed by Rabenhorst in 1902. Almeida classifies piedra as follows: white European piedra (piedra nostras) responsible for nodules on the mustache, rarely the beard, due to *T. beigeli*, *T. ovoides*, and *T. ovale*; white Japanese piedra similar to European piedra and also caused by a special variety of *Trichosporon*; white piedra of the New World (Colombia, Brazil) due to a *Trichosporon*; American black piedra, affecting hairs of the scalp, axilla, and beard and due to *Piedraia hortai*.



Fig. 203



Fig. 204

Figs. 203 and 204 — Nodule of black piedra on hair shaft (Courtesy Dr. E. Muskhelit)

Therapy

Thorough shampooing with tincture of green soap followed by the daily application of a 1:2,000 solution of bichloride of mercury has been recommended. Shaving the scalp first and then applying the bichloride solution or a 3 per cent ammoni-



A



B

Fig 205 —Blastomycosis A Extensive and destructive case involving face and scalp margin (Courtesy Sutton and Sutton Diseases of the Skin Original photograph by Dr J Butler)

B Blastomycosis in an infant (Courtesy Sutton and Sutton Diseases of the Skin Original photograph by Dr J Kessler)



Fig 206 —Coccidioidal granuloma of the scalp (Courtesy Dr F Reiss)

ated mercury ointment is responsible for a more rapid therapeutic success. The Siamese children who allowed their heads to be shaved were rapidly cured and the others responded favorably in time to shampooing with red mercuric iodide soap. A needler attempted to find a solvent for the nodules but was unsuccessful. The author suggests preliminary sponging of the hairs with carbon tetrachloride or ether acetone mixtures to remove some of the more loosely adherent nodules. Some of the wetting agents and emulsifiers should also be effective in loosening the masses.

SYSTEMIC FUNGUS INFECTIONS

Occasionally a systemic fungus infection may involve the scalp. Actinomycosis and blastomycosis may involve the scalp or cranial bones by direct extension, a coccidioidal granuloma may affect it as an acute or chronic process and even sporotrichosis may occur as a primary or disseminated cutaneous nodular lesion on the scalp.

2 ANIMAL PARASITES

Pediculosis Capitis

Pediculosis capitis is the only animal parasitic disease involving the scalp with any degree of frequency. The causative organism is the head louse *Pediculus capitis*. It is about 2 mm long of a rather slender shape and a gray color marked with black spots on the border of the abdominal segments. It inhabits the scalp especially in children of both sexes and in women of questionable cleanliness.

By their bites lice cause severe itching, scratching and excoriations but these symptoms may be altogether absent in some individuals. Without proper care these lesions become infected and the simple crusted papules are superseded by impetiginized yellowish crusts adherent to the hair. In severe cases the patient may develop



Fig 207 Pediculosis capitis. A *Pediculus capitis*. B Oval shaped egg commonly called a nit cemented to a hair. (Courtesy McCarthy Diseases of the Hair)



Fig 208—Alopecia and pyoderma secondary to pediculosis capitis (Courtesy McCarthy Diseases of the Hair)



Fig 209—Impetiginized dermatitis of neck and upper back in association with pediculosis capitis. In all instances of impetigo contagiosa of the face neck or back, the scalp should be examined for pediculi and eggs (Courtesy Dr Frank Combes)

folliculitis and abscesses of the scalp, pyodermatitis and adenitis at the nape of the neck and the lateral cervical regions, and eczematization of the nape of the neck, the ears, and the face. Impetigo contagiosa of the face is often associated with pediculosis capitis, and the scalp should always be examined in these cases.

In a social environment of absolute neglect, the head of some individuals may be found to be covered as with a cap formed by hairs studded with innumerable eggs, teeming with lice, matted together by infected crusts with a nauseating odor, the underlying scalp is bathed in pus. These extreme cases are designated by the names of trichoma and plica polonica.

The deep follicular inflammations and abscesses observed in secondary infected cases of pediculosis capitis may leave a patchy cicatricial alopecia. The secondary pyodermas of the neck have been known to lead to anemia, cachexia, and generalized infections.

The lesions of pediculosis of the scalp begin and predominate in the occipital region—a pruritus or impetigo localized at this point is suggestive of lice and these should be searched for, irrespective of the age or social standing of the patient. If they are not readily discovered on parting the hair, eggs may be found appearing as white or grayish oval grains stuck to the hairs in more or less considerable numbers. The eggs mature in three to fourteen days. They can often be moved up or down the hair shaft (like tight rings on a curtain rod).

Various local remedies are of value in the treatment of pediculosis capitis. First it is necessary to remove all sources of infestation and observe routine measures of cleanliness. Personal combs, brushes, and hats should be disinfested, although the organism is primarily found in the scalp. Pediculicides of value include DDT either in powder form (5 per cent, applied daily for two weeks) or as an alcoholic solution containing benzyl benzoate (10 per cent), benzocaine (2 per cent) and DDT (1 per cent) applied at night, washed out in the morning and reapplied within a week. Hexachlorocyclohexane in ointment form (Kwell) is also effective after one or two applications. The old petroleum cap—consisting of equal parts of kerosene and vegetable oil—is effective when applied overnight to the scalp in turban fashion. Other effective applications include acetic tincture of larkspur (equal parts of xylol and petrolatum lauryl thiocyanate (25 per cent) in petrolatum keep in scalp for ten days) and tincture of *Cocculus indicus* (33 per cent). All applications should be followed by scalp rinses of undiluted vinegar (for two hours) in order to loosen the eggs so that their removal may be completed by means of a fine toothed comb or forceps.

Scabies

This disease rarely involves the adult scalp and in fact is a very unlikely cause of any lesion above the neck line. In infants the itch mite occasionally invades the face and even the scalp (uncommonly). The lesions are papulopustular in appearance and respond to the usual antiscabietic measures.

Onchocerciasis

Another animal parasitic infection of the scalp is onchocerciasis—a filarial disease which is characterized by subcutaneous nodules, ocular involvement and var-

ious cutaneous disorders including elephantiasis. The lesions may also appear on the scalp as firm, elevated, painful, and occasionally suppurative tumors in which the worms (*Onchocerca volvulus*) are enclosed.



Fig 210—Onchocerciasis. These tumors contain microfilariae and are almost always located on the head. (Courtesy Sutton and Sutton, *Diseases of the Skin*. Original photograph by Drs. Strong, Sandground, Bequaert, and Ochoa.)

Loiasis

Loiasis is another filarial disease which may produce similar tumors (calabar swellings) in the scalp. These tumors are grayish white in color with a soft, orange-colored center. The microfilariae may invade the skin itself, producing pruritus, lichenification, eczema, pseudoichthyosis, achromia, and atrophy.

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CHAPTER VI

SCALP DISORDERS OF PSYCHOGENIC ORIGIN (PROVED OR PRESUMPTIVE)

The psychosomatic aspects of scalp disease are many and varied. There is scarcely a disorder involving the scalp in which the psyche fails to play some role. Our everyday conversation is replete with expressions and slang phrases relevant to the scalp and hair fall. Dale Carnegie said, "Just when we're free to enjoy our own leisure, and ought to be happiest, that's when we begin to wonder whether we're getting anywhere in life, whether we're in a rut, whether the boss 'meant anything' by that remark he made today, or whether we're getting bald." The cause of specific types of hair loss may not be psychosomatic, but the latter often becomes of importance subsequent to the development of alopecia. Even in those instances where hair loss is not the bodily expression of psychic tension, it is important for the physician to understand and correct accompanying emotional disturbance. Some form of mental catharsis is often of great value in the treatment of the prolonged and severe types of alopecia. The physician must help the patient to express his fears and worries and bring them out into the open. Reassurance of the disturbed patient with alopecia is often a positive therapy.

The psychosomatic aspects of medical practice are carefully summarized in the Merck manual. According to this presentation,

"Psychosomatic is a relatively new term, coined in an attempt to reunite in medical thinking those inseparable components, psyche and soma that have somehow suffered an artificial dichotomy. In a limited sense, a psychosomatic disorder is one that can be analyzed and understood only when psychologic as well as physiologic factors are taken into consideration. The functions of the nervous system are unitary and integrated, and while we are accustomed to the concept of voluntary and involuntary nerve reactions we are not as yet adequately indoctrinated with the fact that a psychic parallel exists, namely, conscious and unconscious ideation. We accept various derangements of the involuntary nervous system as normal accompaniments of conscious fright and worry, such as examination period anorexia, diarrhea or vomiting. But we are not oriented to the idea that unconscious emotion which by its nature is chronic, may exert long continued similar effects. The fact to be assimilated is that every psychic tendency, i.e. all psychic energy seeks adequate bodily expression.

"Nineteenth century medical thought was dominated by the concept of cellular disease as leading to structural alteration and thus to physiologic or functional disturbance. This idea has become modified in some situations, namely, essential hypertension and vascular disease. Here, it

appears that functional disturbances result in cellular disease and structural change. Now there is a concept that psychologic upset may sometimes antedate and cause functional impairment which, in turn, may progress to pathologic changes in the cells. As yet we do not understand the functioning of our brain as it receives, retains, and later reactivates impulses transmitted to it from the environment, through receptor organs such as the eye and ear. However, that these external impulses result in a more or less permanent functional alteration in the cellular structures within the brain cannot be questioned. To what extent internally originating nervous impulses reaching somatic cells can alter the latter is a question still to be answered.

1 ALOPECIA AREATA

Clinical Features

Hair loss in round or oval patches with sharply defined margins is known as alopecia areata. The seemingly noninflammatory patches may be as small as a few millimeters in diameter or may spread to include the whole scalp, eventually falling into the category of alopecia totalis. The onset of the disease is so rapid that the completely denuded plaque may appear overnight with the lost hairs found in a heap on the patient's pillow in the morning. There are usually no warning subjective symptoms although some persons vaguely recall a slight itching, stinging or burning sensation, while another group complains *post factum* of neuralgic head pains or severe headaches preceding hair fall.

The area which has been freed of all of its hair presents a whitish soft, satiny surface somewhat smooth as compared to the contiguous unaffected skin. In only very rare cases is there a trace of a rosy hue. Pressure on the plaque with the finger often leaves a temporary indentation indicating the presence of slight edema. When pilocarpine is injected, however, the entire scalp sweats except for the involved region where obviously a dysfunction of the sweat glands has developed. Also the application of irritants on the affected portions causes far less reaction than on the normal scalp. At times the follicular ostia are visible on the smooth surface and in rapidly spreading cases minute black points may be seen where the hairs have broken off at skin level or where there is a small heap of cortical cells and granules of pigment the last vestige of the diseased follicles' effort to function normally.

The course of the disease is extremely variable. Either the hair loss is instantaneous or it extends peripherally from a coin-sized patch for two or more weeks until it reaches maximum involvement. It may never progress beyond the boundaries of the original plaque, or several areas may develop simultaneously or at irregular intervals on distant or adjacent sites. The denuded patches may touch at the borders but they never actually coalesce being individually defined. Cases of peripheral expansion however may involve the confluence of several patches.

The circumscribed denuded areas may occur at any site on the scalp. Sabouraud and Lailier found that 50 per cent of their cases occurred in the occipital region. Ciarcocchi reported the same percentage for involvement of the marginal regions as well as on the back of the neck and behind the ears. McCarthy dis-



A



B



C



D

Fig 211 Alopecia areata. A The primary plaque of alopecia areata, regularly oval in outline. B Another primary plaque of alopecia areata showing a fingerlike extent on. More often, a new and distinct plaque makes its appearance. C Another fairly typical primary plaque (courtesy Clay Adams Co). D The amount of hair lost in four months after gradual extension from the primary plaque. (A, B, and D courtesy McCarthy, *Diseases of the Hair*.)

covered that a third of his male patients developed their initial patch in the beard or on the neck but that only 50 per cent of these showed scalp involvement three to four weeks later

In some cases, months and even years may pass without change and then suddenly a new patch may form while the old one has recovered its normal growth. Even old plaques subsequently producing luxurious hair may be reaffected one or more times. Schmidt mentioned nine cases of alopecia areata which reoccurred regularly in September and March of succeeding years, while Smith reported a case of alopecia areata involving the vertex and the center of the scalp which repeated itself every September or October for five years and was of two to three months' duration. The configuration of the hair loss was practically identical each time, and regrowth was complete in the interim.

Walker and Rothman, in their valuable statistical study of this disease, analyzed its course in 120 patients who suffered from alopecia areata from five to thirty six years. The writers were able to follow the course of their involvement for an average of fourteen years. They found that the duration of the initial attack was less than six months for one third of their patients and less than a year for one half. The incidence of relapse was high, being 86 per cent for all the patients observed and 100 per cent in patients observed for twenty years or more. One-third of the 120 never recovered from their initial attack.

The hairs surrounding a bald patch which has reached its natural limits are always normal and therefore not easily removed. If, however, the disease is still in its active phase and engaged in spreading with an ever increasing radius, the long hairs with their glistening root sheaths may be pulled away without effort. Other hairs on the margin of the involved area become fragile and break off, leaving many short hairs from 0.2 to 0.3 cm. in length and of a characteristic shape. On epilation, or after spontaneous fall, one sees that the section of the hair shaft within the follicle is pale, atrophied, and shrunken, terminating in white, pointed bulbs. The nonatrophic, fully pigmented section above the surface of the scalp is considerably broader, giving each hair the appearance of an Indian club or an exclamation point. This is accentuated by the free end being thickened beyond normal, because of a dissociation of the shaft. Hairs of this description may also be found among perfectly normal hairs at the margin of the denuded plaque. They disappear just as soon as the expansion period has been checked and are of prognostic value.

The hairs during regrowth also follow a characteristic course. They first reappear as soft down, the vellus hairs of infancy. This is replaced shortly by a new growth of hair which is thicker and healthier but usually still lacking in pigment, and this crop is further replaced by a dark, entirely normal regrowth. The second stage with its alternating patches of white and dark hair reminds one of the coat of a piebald pony.

Regrowth is not of course inevitable. Many extensive cases seem to be incurable. When the patient has been totally bald for many years beginning with childhood the scalp on examination is found to be so loose that it resembles dermatolysis with its skin and underlying tissue easy to pick up and even to fold

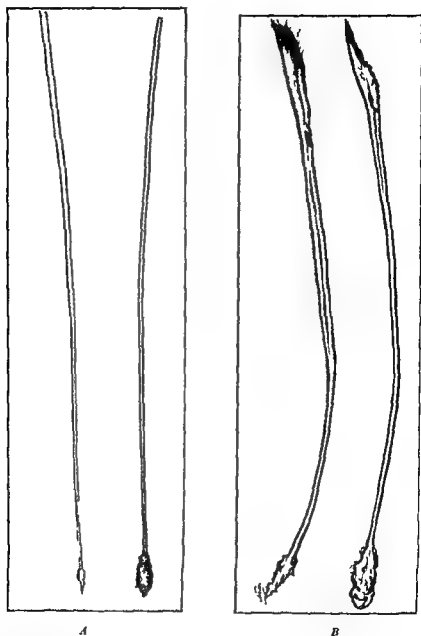


Fig. 212—*Alopecia areata*—typical hairs

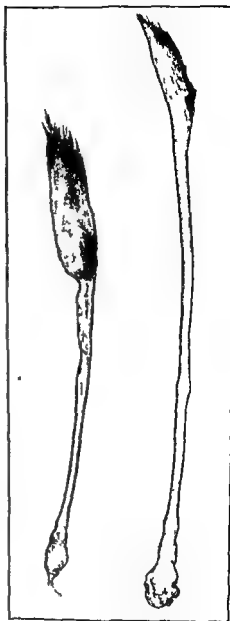
A Atrophic dead hairs (*cheveux caducs* of Sabouraud) The hair to the left has a dry bulb (*bulbe sec*) and the one to the right has a swollen bulb (*bulbe plein*)

B Exclamation point hairs

(Courtesy McCarthy *Diseases of the Hair*)



C



D

Fig 212—C Black point seen occasionally on well-established plaques of alopecia. It is composed of a mass of cortical cells and granular pigment—here still attached to an atrophic hair.

D Cheveux en forme d'épis. Atrophic broken hairs whose acral portion reminded Sabouraud of ears of corn (ép's).

(Courtesy McCarthy. Diseases of the Hair.)

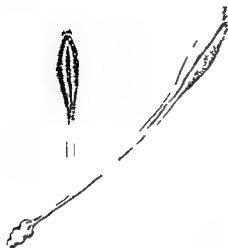


Fig 213

Fig 213 —Drawing of an exclamation point hair from a patient with alopecia areata



Fig 214

Fig 214 —Drawing of cheveux en forme d'eps (ears of corn) from a patient with alopecia areata



A



B

Fig 215 Alopecia areata. A Multiple single and confluent patches. The pigmentation is due to the local application of phenol. B Typical regrowth of white hair in patches of alopecia areata. (Courtesy Dr V. Borghman)

over Alopecia totalis is likely to follow this condition when many patches have developed simultaneously or rapidly following one another, and regrowths tend to fall out again and again. If there is only the extension of one plaque, the prognosis is far more hopeful. If the initial involvement takes place after middle age or is of unusually long standing, atrophy of the hair follicles may occur, resulting in permanent alopecia.

An unusual, symmetrical form of alopecia areata beginning in childhood was observed and described in ancient times as "ophiasis," because of its serpentine character (*ophis* = dim. Greek, snake). A bald area begins at the occiput and progresses in dual bands 5.0 to 7.5 cm wide above each ear, meeting on the brow like a victor's wreath of laurel when the extension is complete. Often the alopecia in these cases is self limiting before encircling the brow. The ophiastic form is rarely cured, for even though regrowth is initiated, there are always successive relapses.

Differential diagnosis is not at all difficult since the clinical appearance and rapidity of onset of alopecia areata are always so characteristic. In its stage of pale colored regrowth, it might be confused with vitiligo except that no history of hair loss is entailed in the latter disease. The various fungus infections of the scalp are easily distinguished from alopecia areata by their usual occurrence in children, the scaling of the patches, brittle and broken hair shafts, and occasional inflammatory changes. No exclamation point hairs are found, and the patches fluoresce a bright green under Wood's filter in the majority of these cases. Microscopic examination of the scales and hairs, plus cultural studies complete the differential findings. "Black dot" ringworm in children due to endothrix *Trichophyton* infection may be reminiscent of the minute black points in the follicular orifices of alopecia areata due to the retention of broken off hairs. When these are epilated and studied, the diagnosis is immediately apparent. Any confusion which may arise from asymmetrical scaly patches of pityriasis steatoides may be dispelled by observing the fatty phases of the disease and by contrasting its very slow and gradual onset without alopecia with the initial overnight hair loss associated with alopecia areata. Early patches due to syphilis are never so sharply defined as those resulting from the disease under discussion (see page 274). The scalp has a mangy, moth eaten appearance and the remaining tufts of hair lack their normal luster. A thorough general examination and serologic studies will eliminate further confusion. In spite of this obvious difference, it is sometimes difficult to reassure patients not that they do not have syphilis but that the patch will not be taken for syphilis by the general public. This morbid fear of public opinion can become so exaggerated that it develops into a trichopathophobia. The follicle free, atrophic patches of the various cicatricial alopecias are easily differentiated from the practically normal smooth skinned areas of alopecia observed in alopecia areata.

Pathology

While the etiologic interpretations of the pathologic findings vary excessively, the sections themselves are not contradictory. Even though erythema is not apparent to the naked eye in alopecia areata, under the microscope there are clearly observable inflammatory changes both in the papillary layer and in the corium.



A



B

Fig 216 — Alopecia totalis

A Total alopecia in a Japanese girl

B Extensive alopecia areata with isolated tufts of hair

(Courtesy Dr E Mandell)

Small round cell infiltration may be observed accompanied by mast cells and larger phagocytic mononuclear types both perifollicular and perivascular. In more advanced cases, the infiltrates are located even in the bands of sclerotic tissue with the sebaceous and lymph glands slightly dilated and coated with this cellular infiltrate, some of which contain thrombi. Levy Franckel, using a capillary microscope, could find no capillaries left in the bald areas and, even after regrowth, very few. Sabouraud and Montgomery investigating many years apart, agreed on the character of the infiltrate, only Giovannini found leucocytes, and Unna reported lymphocytes present as well as mast cells.



Fig 216—C Extensive alopecia areata
D Anterior view of same patient
(Courtesy Dr V Boghosian)

McCarthy calls attention to his findings in sections of early plaques which show a double row of hair in each occupied follicle. While the upper half holds a hair about to be expelled the lower half is attempting to reproduce a new lanugo hair. This inherent desire to regenerate hairs by the follicle is demonstrated by epithelial sprouts and nodes laterally proliferating from the follicular walls. This compulsion to resume its proper physiologic function continues even after the denuded patch has appeared, accounting for the heap of sebaceous material and epithelial cells emptied at the follicular orifices, the minute black points which are sometimes seen on the otherwise white, satiny surface. Bald plaques of several weeks' duration present a thinned epidermis devoid of pigment in the Malpighian layer of the surface epithelium and only one third to one half the number of follicles usually observed on the normal scalp. The regrowth of light or white hairs indicates that pigmentary dysfunction has also affected the pilary apparatus. The follicles that have completely disappeared are replaced by a longitudinal band of sclerotic connective and elastic tissue.



A

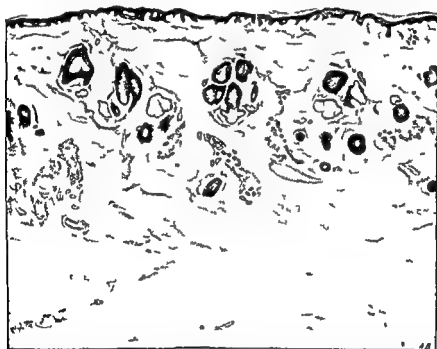


B

Fig. 217. A The marital alopecia type of alopecia areata extending in a zone around the entire scalp. (Courtesy McCarthy. Diseases of the Hair.)

B The alopecia type of alopecia areata with regrowth of white hair. (Courtesy O'Donovan. The Hair.)

From his histologic observations Sabouraud suggested that a deep seated inflammation about the hair papillae interrupts the nutrition of the hair follicles at first interfering with normal growth and later leading to an atrophy both of the follicle and the hair itself. Walker and Rothman believe that the unknown causative factor was responsible for the separation of the hair from its papilla without however, permanently damaging the germinative epithelium. They stress the point that the main pathologic phenomenon is a disturbance of the adherence of the hair to its base and that the site of the attack of the precipitating agent must come at the junction of the germinative epithelium and the hair itself. These writers point out that in its early phase at least alopecia areata resembles postinfectious alopecia and temporary x ray epilation. Of course regrowth in the latter two is entirely normal and no pigmentary change is part of their clinical pictures.



* Fig. 218—Alopecia areata advanced stage. Note the absence of hair follicles with thickened walls and narrowed lumina dilatation of deep vessels and well marked inflammatory infiltrate about follicles and sweat glands. The supporting tissues and glands are apparently normal. The epidermis is atrophic. (Courtesy McCarthy. *Histopathology of Skin Diseases*.)

The demonstrable changes in the hair shaft itself begin with a nodular swelling resulting from the dissociation of the cortical cells and followed by a lessening of the adherence between cells. For these reasons the hair is capable of splitting both longitudinally and laterally eventually breaking transversely perpendicular to its axis at the point of nodular abnormality. The ends of the broken hair are frayed due to the lengthwise splitting presenting the usual appearance of trichorrhexis. The lower part of the hair shrinks in diameter as it atrophies involving the bulb which becomes dry white and pointed.

Etiology

Alopecia areata is found equally in both sexes. Sabouraud reported 130 males out of 200 patients, and Brown's 135 cases included 59 per cent of the male sex. Walker and Rothman's 230 case histories proved equal frequency in both sexes.

Any age may be victimized, but there seems to be a greater incidence in the first half of the life span. Eighty-four per cent of Walker and Rothman's patients developed their bald plaques before the age of forty. Fifty-five per cent of Brown's cases were under twenty-one and, of these, 31 per cent were no older than ten. When the onset occurs before puberty, the course of the disease is more severe, and twice as many in this group eventually develop alopecia totalis. Irrespective of the date of its onset, 75 per cent of all persons suffering from alopecia totalis remain without hair all their lives.

Inheritance as a factor to be considered in its etiology has been discussed by many. Brown could trace the disease to forefathers in 28 of his 135 patients and Sabouraud found 40 out of 200. It often occurs in several members of one family. Hendren recently reported a case of identical alopecia areata in monozygotic twins, 11-year-old boys. Both exhibited a small patch of alopecia areata on identical locations on the right side of the occiput, measuring approximately 2 by 0.75 cm. Below each strikingly similar area there were three smaller regions of thinning hair but no complete loss except in the primary plaque. It is interesting to note that the time of onset was likewise identical.

Walker and Rothman complain of the lack of experimental and statistical study of alopecia areata through June, 1950, and believe that this is the basic reason for lack of knowledge concerning its etiology. Actually, it is true that there is a paucity of collated material from scientific investigations in this field but there is literally no end to the number of case histories whose analysis contributes to an accumulative body of evidence that multiple factors account for the etiology of alopecia areata. Both McCarthy and Montgomery, unable to classify each case under a single, over-all category, assume that the disease is a symptom complex with varying causation but similar clinical features. Sabouraud stated cynically that "every observer can find a proportion of cases to confirm his own particular theory."

The causative factors seem to be, in order of their importance, neurogenic, chogenic, microbic, and toxic. Since more than 50 per cent of all recorded cases show no demonstrable physiologic abnormality whatsoever, it seemed logical to probe deeper into the history of the patient and to uncover causative or precipitating factors that do not meet the eye in the course of routine clinical examination. Robinson and Tasker point out that one important hypothesis which persists through the years is that which links alopecia areata with nervous system dysfunction. Clinical descriptions of such cases during the past hundred years tend to repeat certain characteristic words: "worry," "fatigue," "emotional shock," "anxiety," "fear and mental overstrain." Such terms would seem to indicate that psychogenic factors are more important than physiologic ones.

Fay, in operating to relieve pain in a case of deep carcinoma of the neck and face, severed the sensory and sympathetic nerve supply to the hairy parts of the head without affecting hair growth. Even with the first, second, and third divisions of the trigeminal nerve, the glossopharyngeal, vagus, and posterior roots of the second, third, and fourth cervical nerves no longer functioning on behalf of the scalp, alopecia was still not forthcoming. This would seem to indicate that so far, at least, there is insufficient proof that cranial nerve traumatism is a major factor among human beings.

With his experiments on cats, Max Joseph reported findings to support the trophoneurotic theory. He destroyed the second cervical spinal ganglion, and, from five to twenty seven days later, alopecia simulating human alopecia areata was observable about the ears. Beeson and Pickett repeated Joseph's experiment and obtained like results in only one third of the cats operated upon. Moelli tried a bilateral removal of the spinal ganglion in cats and produced a symmetrical alopecia, while Moskalenko and Ter Gregorianitz recorded like results in dogs as well as cats. These results were flatly denied by Auburn in 1932 who, alone of the experimenters, operated on a sufficient number of cats, 117, to come to any scientific conclusions. After removing both the ganglion and the periphery of the second cervical nerve roots, there were changes noted in only 17 cases. With this limited response, he felt that no specific trophic action was capable of being proved and that probably these instances consisted of partial alopecia and ulceration following trauma due to a hypersensitivity of the affected areas. Wright, also, sectioned the posterior roots of the second cervical nerves of both cats and rabbits and was unable to provoke alopecia. Since no results were forthcoming after all the nerves to the scalp were severed, he suggested that the disease might be due to vasomotor changes rather than to a strictly nervous influence. Schmidt believed that his nine recurrent cases of alopecia areata were the result of a type of arterial spasm due to malnutrition of the terminal arteries.

On the other hand, the hypothesis that alopecia areata is due to functional or psychologic disturbances of the central nervous system is constantly supported by clinical observations and the literature is replete with reports of emotional instability associated with alopecia areata. Psychogenic factors are reported in many cases as the precipitating cause of an attack which subsequently subsided when the emotional problem was solved. There have been many cases coincidental with business failure, the death or departure of a beloved person, and the ordeals of getting married and divorced.

An interesting and somewhat typical case is that of a marine engineer chief of an important ship. While anchored in port, he was off duty and asleep when one of his assistants through negligence allowed the ship's boiler to burst. The chief with great skill and minimum cost, managed a temporary repair job which brought the ship safely into its home port. He expected to be greeted with praise for his engineering ingenuity but instead was suspended from his position because he was held responsible for the character and ability of his subordinates. With more than twenty years of distinguished service on his record, overnight he found himself in a state of disgrace which he considered wholly unjust. An egg

shaped plaque of alopecia areata on the hairy portion of the scalp posterior to the left ear appeared simultaneously. Subsequent clearance of his name did not result in regrowth.

The mechanics involved in psychogenic or functional disturbances of the nervous system in the causation of alopecia areata are probably due to the interruption of normal nerve impulses to the hair papillae. These abnormal impulses are thought to act through the endocrines or sympathetic nervous system with resulting alterations of the nutritional balance of the hair papillae. The association of alopecia areata with neurodermatitis as reported by Robinson and Tasker may be considered additional clinical evidence that a functional disorder of the nervous system is an etiologic factor. Closer observation of patients suffering from either neurodermatitis or alopecia areata may reveal a more usual association of these two dermatologic disorders than has been noted to date. Becker and Obermayer believed that the incidence of nervous trauma is rare in alopecia areata, but Peck seriously doubted whether all their cases were studied to rule out emotional factors in their origin as *not all emotional conflicts are brought to the surface in an ordinary medical interview*. Peck recorded four typical cases which indicate that alopecia areata may be the initial symptom of a generalized neurosis. A 32-year old woman lost a patch of hair 1 mm in diameter. Claustrophobia and nausea in public were frequent concomitant features. Psychotherapeutic interviews ended her neurotic symptoms although the precise significance of hair fall was not explained during the study except that she had a fear of having her hair handled or cut by a barber. A 37 year old male high school teacher with a large family and an obviously emotional and unstable nature, joined the army because of feelings of guilt. Once inducted he was always on sick call. Finally, in North Africa, he was subjected to an air raid during which he went wild with fear, running about the court of the hotel like a madman. He fell and cut his head against a column and by morning was completely bald at the traumatic point. Another soldier, a perfectly stable person was caught in a cellar during a raid. A shell penetrated the shelter, failed to explode rolling and skidding along the floor until it hit the wall. The man was paralyzed with fright and by the next day his hair had fallen out in patches all over his head. Traumatic symptoms of anxiety developed simultaneously and he was subsequently hospitalized. Peck's fourth case involved a feeble minded girl whose alopecia preceded a marked anxiety neurosis.

In summarizing his findings Peck concluded that alopecia areata does appear as one symptom in the opening phase of a neurosis. The first and last were associated with chronic anxiety while the two soldiers suffered onset at experiencing a sudden and, what was for them, an overwhelming fright. In the case of the first soldier, the mechanism seems clearly to be that of a conversion symptom, which may also be the explanation of the second soldier's alopecia since conversion symptoms are frequently met with in the ordinary case of traumatic neurosis and anxiety hysteria. I have seen many similar instances.

Montgomery also postulates that among the middle aged the disease follows obscure disorders of the nervous centers due to unusual excitation whether sudden or prolonged. A large number of patients he admits seem to present cases of in

disputable neuropathic origin. He lists as causative factors blows on the head, nervous shock through fright or great or long lasting anxiety and grief, traumatism of other regions than the scalp, and prolonged confinement in close quarters.

Anderson states that alopecia areata is apparently a reaction of the body to stress, with the hair as a target area and probably occurs in 2 to 3 per cent of the general population. He observed an incidence of 2 per cent among 15,000 hospital outpatients over a five year period. In his series, the most frequent precipitating factor was either mental shock or acute anxiety, which in almost 25 per cent of the cases preceded the onset of alopecia by a few weeks. In 60 per cent of his male patients the initial plaque was in the occipital region whereas in females it was frontovertical.

There are many writers who still present the case of a parasitic agent. Even Walker and Rothman in their statistical report consider the possibility of a microorganism. They point out, as does Montgomery, that those cases which begin as small coin shaped areas of baldness, spreading peripherally in circular lesions, resemble the destructive work of parasites. They present the hypothesis that the microorganism might possibly be one of the many unknown viruses. Epidemics of alopecia areata in schools and orphanages, if correctly diagnosed, would seem to add weight to this etiologic concept. Bowen discovered 63 out of 69 children affected, Fox recorded the incidence among 21 girls between the ages of nine and fourteen, while Davis reported an epidemic of smaller than normal, denuded plaques in an orphanage which involved 174 out of 300 inmates and five months later there were 42 others affected. The 'epidemic' was cured by vigorous antiseptic treatment and isolation. Davis thought it was not parasitic in origin but rather the result of rubbing and mass suggestion. Sabouraud, reviewing these cases of mass occurrence of the disease, decided it was symptomatic alopecia in patches caused and preceded by impetigo.

In the nineteenth century, cultural studies of the hair follicles of the affected areas yielded various micrococci, microbacilli and Welch's *Staphylococcus epidermidis albus*. Vaccines were made from some cultures which seemed to cultivate organisms successfully, but their use proved entirely ineffectual. Sabouraud injecting the microbacillus in calves, rabbits, and guinea pigs, was unable to produce alopecia areata in his animals. All scientific studies have failed to support the contention that a microorganism may even be held partially responsible for this disorder. The argument sometimes advanced that several cases in one family prove the existence of a parasitic agent is invalid. Such occurrences, which are rare, are much more likely to be added evidence of a neurotic or hereditary factor.

In 1932 a different etiologic theory was propounded from Helsingfors by Cederberg. He claimed that he discovered polymorphic spirilla in sections of early cases of alopecia areata stained by the Levaditi process. They were found in colonies within the epithelial tissue and the dermis. They caused a toxicity of the papillae. Cederberg stated with subsequent death of the hair and its follicle. He believed that a spirillum is the main etiologic factor in alopecia areata invading the lymphatics through diseased mucous membranes either in the oral cavity or in any portion of the intestinal tract. A generalized spirillosis followed, with possible

eruptive dermatitis, alopecia and a positive effect on pigmentation. He pointed out that leucoderma and alopecia areata are often found in conjunction on a single scalp. His cultural findings have never been confirmed. Investigators who have carefully performed cultural serologic, and other studies on their patients show a particularly low, and therefore completely incidental percentage of spirochetal infections in their cases. Brown discovered only one positive Wassermann in 60 patients and Orr, only one in 100. Another phenomenon which Cederberg might find it difficult to explain would be the normal pattern of regrowth without specific antispirochetal therapy. Sabouraud studying the ophiasic type of alopecia areata in children found congenital syphilis present in seven out of ten children. It is always possible that alopecia areata may exist in conjunction with other maladies and may even be induced or aggravated by these disease processes.

A third etiologic explanation of alopecia areata is toxicity. Jacquet presented a hypothesis which suggested that this disease is of toxic origin and that with careful search a focal infection may be found associated with either infected teeth, sinuses or prostate. In other words he believed that alopecia areata is merely a symptom of focal disease with, however, a certain predisposition fixed by a disturbed nervous system and an inherited familial tendency to acquire the disease.

Toxicity is not the answer to the etiologic puzzle. In some cases teeth extraction has been effective in most others it has not. Whitfield corrected eye strain with good results. Barber insisted that streptococcal foci in teeth, tonsils, nasopharynx and sinuses were probably responsible while Swill reported a single patient cured after clearing a severe infection of the antrum. There have been several reports of the cure of patients with alopecia areata following removal of foci of infection. I have seen several undisputed instances of cure due to elimination of infection elsewhere. The fact that thallium taken by mouth results in total alopecia of an impermanent nature lent some credence to the theory of toxicity. Unlike alopecia areata the regrowth of hair after thallium epilation is normal; the first crop of new hair does not manifest preliminary changes as is characteristic of alopecia areata.

Two hundred and thirty cases of alopecia areata in the University of Chicago clinic were observed by Walker and Rothman between 1928 and 1948. They concluded after careful study that endocrine factors do not play a causative role. The basal metabolic rates of most patients were normal and clinical examinations failed to reveal any endocrine abnormalities such as disproportionate body structure or fat distribution and unusual genital or mammary development. All functions under endocrine control such as menstruation, pregnancy, lactation and vocal quality were absolutely normal. Warman and Kepler examining 138 cases of alopecia universalis also found no signs of endocrine disturbance. Swill who thought that endocrines might be the chief link in a chain of several causes admitted that her studies of alopecia areata in relation to the metabolic rates of her patients proved nothing. Genner firmly believes in an endocrine factor and lists as his reasons its occurrence in hyperthyroidism in persons with dystrophic nail changes or with vitiligo after shock or trauma in mental disease in persons with disorders of the autonomic nervous systems and the manner in which it disturbs the function of growth and pigmentation of hair.

It is possible that endocrine substances may indirectly modify alopecia areata by conditioning the soil upon which the unknown causative agent acts. Chemical changes in the scalp may occur when precipitated by psychic trauma as a result of nerve impulses and endocrine secretion. Walker and Rothman believe that while the role played by the endocrines, if any, is still impossible to define, a far better case can be built for the hormonal factor as an accessory in the modification of the course of alopecia areata than as the trigger mechanism which sets it off. They call attention to the fact that the course of the disease is more severe when it begins before puberty. Likewise there have been a number of cases of alopecia totalis which recovered completely during one or more pregnancies, only to relapse after menses were re-established. All of these patients, it must be stressed, had perfectly normal general and sexual development, menses, and pregnancies. It is quite possible that the changing hormonal secretions upset the normal physicochemical balance of the unknown mechanisms governing hair growth. If we knew the nature or location of these specific regulatory mechanisms, the mystery of hair growth would be solved.

In a limited number of cases, the onset of alopecia areata coincided with the onset of thyrotoxicosis. Whether cured by x ray, propylthiouracil, or thyroidectomy, the alopecia areata remained. Of Winkler's six cases of alopecia totalis, two showed unmistakable signs of hyperthyroidism. The question naturally arises, what can cause the onset of alopecia areata during thyrotoxicosis and the improvement of its condition during pregnancy? Is there a common denominator to be found? According to Sulzberger, despite some slight evidence of endocrine connections, all old theories are mere speculation and lack satisfactory experimental or factual support.

Therapy

As in all diseases which terminate spontaneously, it is difficult to assess the value of various forms of therapy. All we know is that alopecia areata stubbornly resists treatment, and when a cure has seemingly been effected, it is just as likely that it is due not to the specific therapy employed but to the fact that Nature stepped in at the precise moment when that particular therapy was being employed. Sabouraud, whose experience was manifold, declared that an honest medical man knows that he has not greatly influenced the natural course of the disease. Nevertheless there are certain procedures which should not be ignored and others that should be avoided because they have proved ineffectual. New attempts to solve this therapeutic problem are recorded in recent medical literature.

Approached from the systemic point of view, one might try to perfect the hygienic environment of the patient, prescribe general nutritive measures, treat associated disease, and remove any actual foci of infection. Previous attempts at systemic therapy have proved of no avail. Walker and Rothman report that, at the University of Chicago clinic, therapeutic experiments with alopecia totalis using large doses of estrogen, progesterone, desoxycorticosterone, and lactogenic hormones were unsuccessful except for one case whose course was merely arrested by administration of the latter. Histamine and similar agents administered by iontophoresis cause marked vascular dilation and produce an effect on general circulation but none

whatsoever on the alopecia areata. On the other hand, Gougerot and associates also believing that sympathetic vasoconstriction played the most important role in alopecia areata, injected methylacetylcholine intradermally, thus causing vascular dilation for at least an hour at a time. These injections were given twice weekly in doses of 0.05 cc (solution containing 4 mg to the cubic centimeter) with supposedly good results. A plaque with a diameter of 8 to 10 cm required three to four injections, according to these observers.

Approached from the viewpoint of local treatment there are many favorite remedies all of which frequently prove useless. Savill, nevertheless, believes that any method which evokes local inflammation has some value, particularly in the chronic cases which should be kept in a continuous state of irritation. Therefore any stimulating lotions or ointments and antiparasitic preparations may be used alone or in combination followed by oil or a simple ointment. Ormsby and Montgomery list at least 19 possibilities including alcohol, ether, formaldehyde, camphor, phenol, tar, iodine, sulfur, the mercurials and many others. Sabouraud preferred oil of cade mixed in a base of equal parts of petrolatum and lanolin, applied at night and removed the following morning with acetone or ether. Savill tested this preparation on half of an extensively involved scalp using her own favorite as a control on the other half (a combination of equal parts of sulfur and of iodine). She found them equally successful.

I prefer the local application of undiluted phenol with neutralization by alcohol as soon as or just before blanching appears. This application is alternated weekly with suberythema doses of ultraviolet radiation from a mercury quartz burner. The patient is also given one of the lotions or salves listed in the Formulary (page 347) for daily home use. Inasmuch as these applications are frequently irritating or sensitizing in their action the patient should be kept under observation.

The newest attempt to find a method of local treatment which would be efficacious was reported by Feeny. He treated twenty-four cases of alopecia areata including three cases of alopecia totalis with monthly applications of thorium X in varnish from a few weeks to a year. He carefully excluded from this series all patients whose improvement might have been attributed to concurrent somatic or psychologic treatment. Twenty-two were successful, two were not. Ten patients had had alopecia areata for over ten years and nine of these had had local applications of light from a few months to two years. Regrowth started in two months for five out of the nine, in four months for two others and in eight months for the last two. The cases of total alopecia responded with partial regrowth which was normally pigmented. The entire process according to this observer was superior to results reported from the application of the ultraviolet light besides taking less time. Feeny warns that the applications should never be used more than once a month. Confirmation of these results has not been reported and in my opinion should not be expected. There has never been any proof of the statement that radiation, whether of a soft alpha or heavy gamma type, has any stimulating properties as far as hair growth is concerned. If anything the effects of such forms of radiation are depressing or destructive to the cellular components responsible for the physiologic growth of hair.

In all patients whose histories indicate a psychogenic etiology, a psychiatrist or analyst should be called in consultation and, when so indicated, psychiatric therapy should be employed. Whatever neurosis or emotional conflict the patient may suffer from, its eventual solution may directly affect the course of alopecia areata, although such conflicts are neither constantly present nor necessarily of fundamental etiologic import. The psychotherapeutic measures described under the heading of Circumscribed Neurodermatitis, Therapy, page 384, are also of value. Recent reports citing the efficacy of cortisone require further confirmation.

2. CIRCUMSCRIBED NEURODERMATITIS

(SUBOCCIPITAL DERMATITIS; LICHEN CHRONICUS SIMPLEX)

Clinical Features

Intermittent and intense itching of the scalp without any skin change other than a dark pinkish tint heralds the approach of suboccipital neurodermatitis. Due to persistent scratching and rubbing, this area finally develops into a slightly raised patch of thickened skin the normal markings of which are grossly exaggerated.



Fig. 219—Small nuchal patch of neurodermatitis, localized over occiput in the hairy scalp.

These plaques are most often nuchal in origin and may be oval or angular in shape and of widely varying size, but they usually present the gridded appearance of lichenified skin with its rectangular papulelike lesions. The base of each patch is



Fig 220 — Neurodermatitis *A* Neurodermatitis of the ears and retroauricular folds with pityriasis amiantacea of the scalp *B* Right side of scalp *C*, Left side of scalp (Courtesy Dr W Herbert Brown, Glasgow)

erythematous and sometimes involves an area larger than that of the initial lesion thus forming a reddened periphery. Thin branny scales or, in chronic cases thick psoriasiform scales cover the surface with no bleeding apparent upon their removal. On palpation there is no yielding of the scalp but rather a rigidity reminiscent of an early stage of morphea.

Itching is acute occurring in paroxysms and crusts often follow involuntary scratching. While there is no actual hair fall this persistent irritation by the patient sometimes accounts for the fracture of hairs (trichorrhexis) at the skin's surface. Beginning at the nape of the neck in the hairy margin of the scalp the neurodermatitis spreads both up and down and at times involves regions as far forward as the ears. In Lynch's study of 83 cases of this disease, he found that variation in the size of the plaque was practically limited to the nuchal fossa and its



Fig. 291.—Neurodermatitis involving the ear and auricular skin. Typical patches were present elsewhere on the body and on the vertex of the scalp. (Courtesy Dr. W. Herbert Brown, Glasgow.)

margins; in other words the involvement was chiefly from 1 to 2 inches in diameter and rarely unilateral. In addition to affecting the scalp and neck, similar plaques are often situated on the cubital and popliteal regions as well as on the eyelids, thighs, ankles, and dorsal aspects of the feet and forearms, although the eruption may be limited to the nuchal region alone.

Brocq pointed out that each circumscribed patch could be divided for minute descriptive purposes into three clearly defined zones: The center is composed of

an area of lichenified, infiltrated skin as noted above, red at its inception and dark brown as it ages. Around this there is another, either rosy red or grayish, made up of a great number of flat, shiny surfaced papules, confluent toward the center and discrete toward the outer zone. The latter or third zone is from 1 to 3 cm in width, covered with many very small, slightly elevated papules resembling the nap of a coarse brown or café au lait plush.



Fig. 272—Neurodermatitis of the nuchal region with localized alopecia and typical patches elsewhere on the body. (Courtesy, Dr. W. Herbert Brown, Glasgow.)

There are two varieties of neurodermatitis: the circumscribed lichenoid or localized and the diffuse or generalized (prurigo, atopic eczema). The diffuse type involves greater areas of the body and scalp surface. Pruritus is equally intense and precedes the onset of the discolored and thickened, crusted papulelike lesions which are irregularly distributed over the regions affected. This type affects younger individuals, is less likely to form large lichenified plaques, and the atopic factors are of far greater significance. Chronic circumscribed neurodermatitis of the neck or scalp usually is limited to that location and appears as a single lichenified, sharply margined plaque. This characteristic appearance and the restriction

of the lesion to a single site, as well as the absence of the various manifestations of allergic disease, serve to distinguish this disorder from atopic dermatitis. However, in a fair percentage of cases, it is actually only one of the usual sites involved by that disorder. Alopecia is not as uncommon in this disease as is usually believed.

Differential diagnosis is simplified by the characteristic and individual site of the initial lichenoid, sharply demarcated plaque, by the extreme intensity of the itching, the great accentuation of skin markings, and the resistance to local applications which normally alleviate the discomfort suffered from eczematous or seborrheic dermatitis. The latter does not produce severe itching and the scales are



Fig. 223—Extensive neurodermatitis and diffuse alopecia of the scalp. The clinical features were those of pityriasis amiantacea. (Courtesy Dr. W. Herbert Brown and Brit. J. Dermat.)

greasy. In the former the process is more likely to be acute and vesicobullous in nature. It is of fundamental importance that the possibility of contact dermatitis be eliminated before making a conclusive diagnosis of neurodermatitis. After neurodermatitis has persisted for some time and the scales have become psoriatic in aspect no confusion need usually exist because psoriasis is not accompanied by acute pruritus and when its scales are lifted a bleeding surface is disclosed which is totally absent in neurodermatitis. However, both diseases may coexist. In the disease under discussion once called lichen chronicus simplex the better defined plaques, their rosier hue, and shiny flat topped papules distinguish it from lichen planus. At times there

may be a case which superficially resembles papular eczema, but the latter's small red, acuminate papules do not parallel the numerous little squares and rectangles formed by the crisscross wrinkles of a lichenified neurodermatitis. Alopecia areata may coexist with neurodermatitis.

Pathology

Ormsby and Montgomery found both hyperkeratosis and spotty parakeratosis usually accompanied by papillomatosis and irregular acanthosis. In certain cases, the acanthosis may be so well developed that it produces verrucous lesions. In the early stages, the upper cutis is normally infiltrated with a moderate amount of lymphocytes and eosinophiles. Later the infiltrate is either perivascular or diffuse, but never perifollicular.

In 1949 Lynch performed 29 biopsies in his investigation of suboccipital dermatitis to make sure that it was actually neurodermatitis and not a separate entity. He reported that while hyperkeratosis was rather often present, it could not be called either constant in occurrence or striking in degree. He found that parakeratosis was constant, not pronounced, and usually localized to a comparatively small area. When parakeratosis was present, the stratum granulosum was absent, but in other cases it was perfectly normal.

Lynch found a moderate amount of acanthosis consistently present and often associated with elongation of the rete pegs. There was frequent degeneration of individual cells or groups of cells in the rete layer. The rather small invading lymphocytes caused an increase in the edema of the infiltrated areas accounting for the development of minute vesicles, although these vesicles have not been reported by other observers. Unlike Ormsby and Montgomery, Lynch discovered these only singly or in groups of two or three, never with epithelial abscesses. He also disagreed as to the amount of eosinophiles present. They appeared only rarely as did the incidence of red blood cells. Epithelial edema was common, but only from a slight to moderate degree and only 16 per cent demonstrated any changes even resembling vesiculation.

While Ormsby and Montgomery found the elastic and connective tissues as a rule untouched, they did report an increased amount of melanin in the basal and lower layers of the epidermal cells in lesions which had persisted for some time. Lynch noted that he found many connective tissue changes not seen by Ormsby and Montgomery. This tissue often appeared swollen and fragmented. Special stains also showed the elastic tissue in process of degeneration and fragmentation even when the lesion was comparatively new. Capillary dilatation was always present. Ten of the twenty-nine biopsy specimens showed a thickening of the walls of the larger vessels.

Pathologic findings also assist differential diagnosis. Although the rete ridges are elongated, they are not inclined to be clumped as in psoriasis. Also the abnormally twisted course or apparent rigidity of the capillaries as seen in psoriasis is totally absent in neurodermatitis.

Recent histologic observations by Laymon of sections from patients with dermatitis of the nuchal region showed considerable variation. Hyperkeratosis al

though present in most sections, varied a great deal both in degree and regularity. In some cases it was mild but uniform, in others moderate to intense but uniform, and in still others mild and spotty. Parakeratosis was present in every section but one, and appeared uniform in some and localized to small areas in others. The stratum granulosum was present in most sections although as a rule it seemed thinner than normal. Acanthosis was present in all his cases but one, in which there was a surprising epidermal atrophy. As a rule, the acanthosis was moderate. In some sections it was uniform, although in most of them it was irregular. In most instances the rete pegs were elongated, although no clubbing was noted. Slight to moderate epidermal edema was usually present. He noted no evidence of vesiculation in any of his cases, and no microabscesses were seen. Mitosis in the epidermis was so insignificant that it seemed to be of no diagnostic value. There was nothing noteworthy as far as the basal layer was concerned. In about 25 per cent of the cases, crusting was evident, as characterized by accumulation of debris and polymorphonuclear leucocytes on the surface. In all probability this reaction was a result of trauma following scratching.

According to Laymon, the features in the cutis were nonspecific. Mild edema was present in most instances along with moderate dilatation of the smaller vessels. The cellular infiltrate was usually located in the upper portion of the cutis about the vessels and ranged from mild to moderate in intensity. There was no tendency toward arrangement about follicles or glands. The infiltrate was almost exclusively lymphocytic, although occasionally polymorphonuclear leucocytes and eosinophils or red blood cells could be seen.

In 1946 Sachs, Miller, and Gray discussed the "neurodermatitic reaction," a term not accepted by many dermatologists, which has been used to represent the pathologic process rather than a disease and applied to such dermatoses as neurodermatitis, nummular eczema and exudative lichenoid and discoid chronic dermatitis. These authors stated

"The pathologic process involves both the epidermis and the cutis. The former is dry and, although the intercellular spaces may be accentuated the basal cell margin is unaltered, the granular and horny layers are present and may be increased, and a rather regular acanthosis is usually present. However, friction, scratching medication, etc., may superimpose a traumatic dermatitis. This would account for the presence of an irregular acanthosis, edema and even parakeratosis."

They felt, also, that the changes in the cutis were nonspecific and concluded that the pathologic observations could not establish a final diagnosis.

Lynch in his recent discussion of suboccipital dermatitis, described similar changes and brought out the difficulties in the histologic diagnosis of neurodermatitis, psoriasis, and seborrheic dermatitis. He concluded, after a review of the microscopic changes, that histologic examination was of no great aid in differential diagnosis. Laymon's studies confirmed these opinions.

Engman and his co-workers found by microincineration and spectrographic analysis that the epidermis of the normal skin contains a relatively large amount of calcium and magnesium. Epidermal cells of patients with neurodermatitis con-

tained little of these salts. Biopsy examination of later specimens of spinous cells from the active lesion of neurodermatitis do not show retention of calcium nor magnesium but healed lesions regain calcium in large quantities. One hundred and six out of 176 cases tested showed a definite calcium deficiency according to these observers.

Etiology

The variety of skin involvement is the direct result of the patient's inability to control his scratching of pruritic regions of his scalp by day and especially by night when his controls are at the mercy of his subconscious. The habit then becomes a form of psychologic relief from worry and tension.

The chief incidence seems to be in women over 25 years of age. In Ormsby and Montgomery's series of 450 cases the ratio was 2:1 in favor of the female sex and the majority of those were over 40 years of age. Lynch found in his cases a lower incidence in the group under twenty and a higher incidence in the decade from thirty to forty. When suboccipital dermatitis is compared with neurodermatitis of other parts of the body the most evident variation is the increased incidence of its association with abnormal menstruation. The nature of the abnormality is still vague but it seems in most cases to indicate an estrogenic deficiency. Lynch found that 27 per cent of the women under twenty with suboccipital dermatitis and 45 per cent of those between the ages of twenty and forty suffered from abnormal menses. Of those suffering from disseminated neurodermatitis only 17 per cent under twenty and 27 per cent of those between twenty and forty had the additional difficulty. He therefore concluded that abnormal menses occurs much more frequently among patients with suboccipital neurodermatitis than in general and that there is a significant connection between suboccipital dermatitis and ovarian dysfunction.

The affected women are usually high strung and of a temperament described as nervous. Scarborough discovered that male patients suffering from neurodermatitis seem also to be sexually maladjusted, suffer stronger feelings of hostility, fear and guilt, have had childhood and adult masturbation conflicts and in most instances derive some secondary benefit or gratification from their illness. Even after their conflicts have been resolved and their repressions brought to the surface by psychotherapy, these patients require a certain amount of continuous psychiatric guidance.

For many years the atopic background and allergic reactivities of these patients have been stressed. Many authorities consider the disease due to hypersensitivity to ingested, inhaled or contacted allergens in individuals with an abnormally high familial and personal history of atopic disease. Although this is often true the etiologic importance and therapeutic value of such associations have been overemphasized in my opinion. It is probable that the pathogenetic mechanism is as follows: given an individual with an atopic background the emotional forces act as the labile fuel feeding the pruritic fires. Wise stated in his study that asthma, hay fever, and bronchitis bear some relationship to neurodermatitis. Lynch questioned his patients about other atopic disorders such as hay fever, asthma and childhood

eczema and discovered that 62 per cent of the suboccipital dermatitis group had such a history of atopy and 75 per cent of the group suffered from associated neurodermatitis in addition to the suboccipital variety. In the special age grouping of those over forty, 91 per cent had such a history. This is usually considered as adequate proof of the atopic and allergic nature of these diseases. However, these associations must not be accepted as definitive inasmuch as hay fever and asthma are thought to have at least a partial etiology in the realm of psychosomatic disorders. The proper delineation of these diseases and their causative mechanisms still require clarification for their eventual denouement.

Lynch became interested in discovering whether suboccipital dermatitis had any important characteristics different from those of neurodermatitis in general. He wondered whether a study of the site would be revealing as concerned the reasons for the endocrine implications noted above. Neither specific allergic reactivity nor seborrheic or infectious factors seemed capable of explaining the selectivity of location. External factors such as heat perspiration and production of sebum if responsible would have to be activated by autonomic nervous mechanisms. Endocrine agents however often manifest a selectivity in the location of symptoms sometimes influenced by anatomic structure and other times by means of vasomotor phenomena. Psychologic and emotional deviations often modify the site of disturbed function also by way of the autonomic system.

The mechanism herein is not completely understood but it is of interest that women frequently suffer from a nuchal feeling of tension during the climacteric that a dog's favorite site for stroking is in the region of the occipital protuberance that a cat also finds erotic satisfaction in the same region and that many a human being has been known to purr at the tender stroking of this same hypersensitive area.

Language and most particularly slang expressions can sometimes throw a searchlight into the darkness of the seemingly inexplicable as Freud so brilliantly demonstrated. Lynch calls our attention to the well known fact that many incidents which lead to psychologic dissatisfaction or persons who aggravate us are said to be a pain in the neck.¹

Therapy

There are three approaches to alleviating the suffering caused by neurodermatitis: local, systemic and psychic. Antipruritic agents such as phenol, tar, menthol and camphor afford temporary relief. Effective preparations for use on the scalp are listed under the section on Pruritus (page 403). Crude coal tar ointment is preferred by Savill but the patient finds its odor disagreeable and its adhesiveness annoying. Sabouraud recommended 1 to 2 per cent iodine in alcohol followed by a massage with 1 to 6 per cent coal tar ointment which could later be removed by cotton soaked in equal parts of acetone, alcohol and distilled water. McCarthy suggested painting the lesions with a 5 per cent solution of silver nitrate twice daily for several successive days to relieve itching. Coers used five weekly applications of thorium X in varnish (2,000 units to the cubic centimeter) painted on the affected region with good results. Exposure to roentgen rays may be of value but the

amount must be carefully controlled (1×75 r, at weekly intervals, with a low voltage technique) so that it will not cause epilation. Grenz rays have their proponents. I rely locally on the following applications:

Vioform or Diodoquin	30
Liquor carbonis detergens	10
Ianolin	150
Petrolatum	qs 1000
Phenol liquef	10
Pine tar ointment	150
Zinc oxide	150
Cold cream, unscented	qs 1000
Sulfur ppt	
Oil of cade	—
Salicylic acid	as 30
Aquaphor	qs 1000
Menthol liquef	02
Antihistamine powder	10-50
Water washable cream	qs 1000
Vioform	10-30
Antihistamine cream	qs 1000
Liquor carbonis detergens	as is
Sig. Apply at night and remove with vegetable oil in morning	

An occasional resistant patch may respond to two weeks of daily paintings of crude coal tar or liquor carbonis detergens followed by a short course of x-ray therapy as described above. I have also treated small patches with applications of 10 per cent potassium hydroxide and of 50 per cent trichloroacetic acid, thereby substituting burning and pain for itching, surprisingly enough preferred by the majority of patients.

The general activity of these patients should be controlled as much as possible by the physician. The use of stimulants should be discouraged and a carefully balanced program of work and recreation should be planned.

Stokes has outlined an admirable group of measures which he designates as a basic, psychotherapeutic approach to the patient with eczema, hay fever, or asthma. These measures are as follows:

"For the infant and prepuberal child

- "1 Supervised play with other children under child guidance and nursery school direction
- "2 Calling off of the oversolicitous and overprotective parent
- "3 Treatment of the child as an equal, with explanation, patience, laissez faire and persuasion replacing to varying degrees parental sovereignty, severity, repression, irritability and 'don'ting'
- "4 Elimination of as many contacts as possible with the irritating ('electric') or irritable and repressive parent or other personality (in school or away from home)

- 5 Adjustment of marital and personality conflicts between parents and insecurity producing discriminations among children in the family, as promptly and early in life (even prenatally) as possible
- 6 Discouragement of the forcing tactics marks, standards and competitive techniques of teachers schools and sports
- 7 An attempt to manage the parent problem as in the child but more circumspectly
- 8 An effort to identify in simple conversations on current activities of the patient his plans and problems the more obvious conflict factors and especially the critical inferiorities which in crisis form can precipitate asthmatic and neurodermatitic explosions
- 9 Education as to I sensitiveness kinetic drive and effect of competition in one or two simple talks
- 5 Systematic encouragement combined with emphasis on self acceptance and depersonalization of outlook
- 6 An attempt to manage the parent problem as in the child but more circumspectly
- 7 An effort to identify in simple conversations on current activities of the patient his plans and problems the more obvious conflict factors and especially the critical inferiorities which in crisis form can precipitate asthmatic and neurodermatitic explosions
- 9 Training in self confidence and self reliance (exploration experiment, trip alone, direct relations with teachers advisers and physicians not mediated through the parent and systematic but not exaggerated encouragement)

For the adult

- 1 Not too much explanation of the mechanism which characterizes or creates the mental state at least not to the more unstable subjects
- 2 An attempt to manage the parent problem as in the child but more circumspectly
- 3 An effort to identify in simple conversations on current activities of the patient his plans and problems the more obvious conflict factors and especially the critical inferiorities which in crisis form can precipitate asthmatic and neurodermatitic explosions
- 4 Education as to I sensitiveness kinetic drive and effect of competition in one or two simple talks
- 5 Systematic encouragement combined with emphasis on self acceptance and depersonalization of outlook
- 6 An attempt to manage the parent problem as in the child but more circumspectly
- 7 An effort to identify in simple conversations on current activities of the patient his plans and problems the more obvious conflict factors and especially the critical inferiorities which in crisis form can precipitate asthmatic and neurodermatitic explosions
- 8 Training in self confidence and self reliance (exploration experiment, trip alone, direct relations with teachers advisers and physicians not mediated through the parent and systematic but not exaggerated encouragement)
- such as rowing canoeing swimming sailing skiing or skating—not driving a car which is a poisonous joy for persons of this kind
- 9 Change of scene (i.e. adjustment by flight) only in crucial and unmanageable situations

Stokes also lists the following measures to be employed in the basic psychotherapy of the patient with the tension personality

- 1 A talk on general principles in which the fundamental nature of tension is explained with its relation to behavior of the skin
- 2 The attack on the obligatory in repeated sessions by the injunction *not to do whatever gives the patient a feeling that it must be done*
- 3 Cutting off access of the most influences to the patient (in severe cases no mail no answering of telephone no making or carrying out of engagements no acceptance of new social obligations and a repudiation of up to 50 per cent of current social commitments)
- 4 Provision of physical sanctuary (i.e. own workroom or garden) to be used for a set period each day
- 5 A lecture on the don't give a damn attitude—or temporarily conscienceless attitude—illuminated by such slogans as a hundred years from now

- 6 Drill in relaxation, both systematized as in exercises, and waiting with patience as in doctors' offices or for traffic lights substituting shrugs for jaw clenching and nail gnawing
- 7 Provision for discharge of tension by noncompetitive physical means (notably again walking) manual hobbies and stopping of competitive sport 'exercise'
- 8 An arbitrary rest prescription based on the preceding elements, usually a one hour nap or a nap plus relaxation (with perhaps graded sun bathing), following if possible by one hour of a hobby, in sanctuaries)
- 9 Conversational exploration of simpler conflicts (often part of the history taking) with suggestions for adjustment
- 10 Systematic practice in living for the moment and viewing life with serene detachment (the big and little Mary' device helps)
- 11 Systematic encouragement with practice in self esteem and self acceptance
- 12 Education of the patient in the recognition of his own signs of tension including watching his own voice, hand eye and forehead and in the application of remedies"

Heinhaber has reported some success in the relief of neurodermatitis by the use of procaine infusions or of a local anesthetic such as Avertin. Patients are hospitalized and the treatment is administered under the care of a trained anesthetist. In my personal experience the relief obtained by this method is only temporary. Two of Heinhaber's patients were without the uncontrollable pruritus, loss of sleep and the attendant anxiety for six and nine months, respectively. Others however, had respite as brief as two weeks.

When everything possible has been done to relieve the pruritus locally and systemically and a basic disturbance of the nervous system is still finding its outlet in itching and scratching, psychotherapy should not be neglected.

3 TRICHOTILLOMANIA

Clinical Features

A rare form of partial alopecia is the result of trichotillomania, the neurotic compulsion to pull out one's own hair. The areas of the scalp denuded by this process are usually those most accessible to the right hand of the patient. They are therefore limited in extent, never involving more than a strictly circumscribed region, usually behind the ear on the temples or above the forehead. Often the eyebrows or moustache have been similarly attacked.

The plaques differ from those precipitated by other causative factors in their shapes, which are either too regularly patterned or too irregularly without clear definition. Instead there are often no exact borders and the denuded area may be described as merging into the encircling hair growth.

The scalp which has been exposed in this fashion is usually perfectly normal unless the patient also complains of pruritus in which case it may be red and show the excoriations of scratching. The alopecia is never complete. On the bald patch it is always easy to locate healthy hairs which were too young and therefore too short to be pulled out. On old areas where regrowth has been permitted to start there are normal hairs of varying lengths representing different age levels and these resist epilation to the same degree as all healthy hair.

The follicular orifices are often filled with black points, making them clearly visible. Some are the result of heaped up cortical cells being pushed to the surface by the next hair developing within the follicle. Others are hair shafts which were left behind when the hair, instead of being cleanly epilated, was broken off at the surface of the skin. This latter technique belongs to a slightly different neurotic compulsion known as trichokryptomania or trichoclasmania which is often found in association with trichotillomania.

There are seldom any subjective symptoms, the compulsion needing no local precipitating factor. Some patients, however, claim that they began the practice because of an initial itching sensation, with the forced epilation providing a temporary salutary counterirritant.

Differential diagnosis is not at all difficult. The lesions are so bizarre and artificial in appearance, their sites so characteristically located on the scalp, which itself shows no signs of abnormality, that the clinical picture is not at all confusing. The short, healthy hairs which escaped epilation are always present, while there is no sign of any exclamation point hairs. The patient himself confirms the diagnosis by being a person of a highly nervous type or even in extreme cases mentally deranged. Sutton describes them as always exhibiting symptoms of neurocirculatory instability.

Etiology

The tic of depilation, a neurotic compulsion, was first named trichotillomania by Hallopeau, who originally thought that it was the result of intense pruritus of the scalp. Since his hypothesis it has been reported most frequently independent of any itching, burning or tingling sensations. The infant in his crib or nursing at his mother's breast twists his hairs about his fingers and pulls, demonstrating that this behavior is fundamentally one of a perverse means of sexual gratification. The adult who indulges in this neurotic behavior is producing pleasurable pain.

English, in his analysis, asserts that such persons lack more natural and whole some means of relieving tension. Their frustration results from their not having been allowed to find normal outlets or not having been taught what these are early enough in life. The habit which they form of forcibly extracting their own hairs he believes relieves the momentary tension at the same time as it produces a sensation which they find enjoyable. Although an uncontrollable habit of such a nature obviously puts them at a disadvantage in the eyes of the world, they have an insistent urge to repeat the process for the pleasure which it gives them. It is a true mania since it is an impulse which defies self control.

McCarthy described the case of a woman who indulged in trichotillomania only during her pregnancies. Forty-eight hours after each birth she had no difficulty whatsoever in terminating her formerly compulsive behavior.

Therapy

Psychotherapy is obviously the most important therapeutic measures to be considered in the management of trichotillomania. For the insane, institutional care



A

Fig. 224—A. Trichotillomania with the oftentimes associated excoriations of the scalp. (Courtesy Dr. Frank Conley.)



B



C

Fig 224 B and C

B Vitiligo and poliosis in association with neurodermatitis (Courtesy Dr F Reiss)

C Alopecia areata in association with neurodermatitis (Courtesy Drs S Robinson and S Tasker)



Fig 225 - Extensive trichotillomania in a 16 year-old girl. Note irregular outlines of lesion and different lengths of broken hairs. (Courtesy McCarthy. Diseases of the Hair.)



A



B

FIG 226 --A Trichotillomania in psychotic individual B Self induced eruption, same individual (Courtesy McCarthy D waves of the Hair)

is essential but for the otherwise normal patient psychiatric or psychoanalytic interviews invariably assist them to control their impulse. English points out that it is the duty of the physician to help his patient endure tension on the cessation of the habit channeling his energies into more wholesome and useful directions. If the trichotillomania is a neurosis with a pathologic base and it has been precipitated by some trivial occurrence the habit can be stopped if the dominant obsession can be uncovered.

Local measures recommended by some writers have only a superficial effect. The suggestions that the hair be kept too short to pull out or that it be totally removed for six weeks by x-ray epilation to break the rhythm of compulsion are at best temporary stopgaps. While the frustration exists the neurotic symptoms will persist.

4 ACAROPHOBIA

Clinical Features

This is a mental affliction in which the patient honestly believes that his scalp and other regions of his body are infested with crawling insects. This intensely irritating sensation persists even though the physician demonstrates that no living organism has adopted his integument for a home. In addition to the creeping and crawling sensations sometimes an acute pruritus is present. Either the skin shows no change whatsoever or it gives evidence of traumatic dermatitis brought about by the sufferer's attempts to dig out the offending insect and so free himself from his maddening symptoms. These typical neurotic excoriations are usually no larger than 0.5 cm in diameter, often covered with bloody crusts and usually presenting an erythematous periphery. When the crust falls off a pale pink scar with a hyperpigmented border remains.

In their anxiety to make the physician believe in the reality of their supposed infestation the patients often bring small particles they have rubbed off or dug out of their own skin and which they believe are vestiges of the dreaded insect life. Even though the doctor assures them that this is merely normal epidermal debris or even particles of lint from their own clothing acceptance of reality is impossible for the patient. There can be no confusion between acarophobia and follicular dermatitis because the patient with the former disease is only too willing to admit that the lesions on his scalp and body are self-inflicted.

Etiology

Sabouraud's experience led him to believe that the majority of such cases came as a result of taking cocaine or heroin. He recommended that the physician carefully search for the puncture marks of the hypodermic needle before attempting other forms of therapy. Case histories in countries other than France show that very few indeed were incited by drug addiction.

In Lewis and Cormia's classification of varieties of neurodermatoses they have placed acarophobia in the category of dermatitides which are always psychic in origin. Wright in his article on psychosomatic dermatoses places delusion of parasitosis at the top of the list of purely psychic phenomena. Involutional melancholia

has been suggested as a psychic causal factor, but the whole conception of insects associated with filth, and filth associated with immorality or degeneracy, seems to connect acarophobia with other manifestations of a deeply felt guilt complex

Therapy

The only therapy is psychoanalysis

■ PITYRIASIS AMIANTACEA (TINEA AMIANTACEA)

Clinical Features

This disease is characterized by the gradual appearance on the scalp of an extremely thick layer of silvery white or dull gray scales. The scales are laid one on top of the other, like shingles on a roof. They cling in a mass to the scalp and

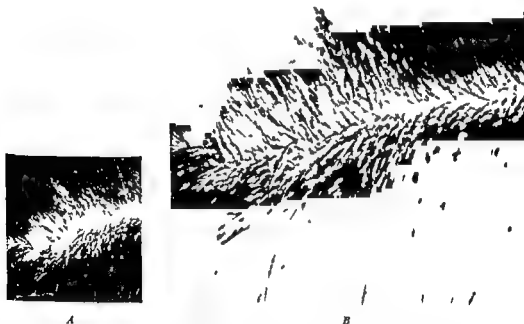


Fig. 227—Pityriasis amiantacea. A A typical patch in the scalp of a patient with neurodermatitis of the scalp, ears and antecubital regions. B Enlargement of the area shown in A. (Courtesy Dr. W. Herbert Brown, Glasgow.)

hang onto the hairs as an asbestos-like layer for some distance along their proximal portions. Many different views have been expressed as to the cause and method of production of these asbestos-like scales. Most authors agree with Sabouraud and McCarthy that the disease shows two distinct stages and represents a type of eczema of the scalp. It begins with a moist stage that results in constant "weeping" of the scalp. This secretion gives the scalp a peculiar and offensive odor. This exuda-

tive scalp surface appears reddish or reddish yellow and considerably swollen. The excessive secretion pastes the hairs together and glues the whole mass to the surface of the scalp. Miliary pustules or impetiginous vesicles may suddenly scatter themselves over the inflamed areas. In many cases this primary weeping stage may have been passed through before the patient is seen by the physician. However, clues of the acuteness of the process can usually be detected during this early stage if searched for under the mass of scales.

The second or 'dry' stage is characterized by the deposits of peculiar, layered dull gray or gleaming white scales along the hair shafts. These scales cling to the hairs in an asbestos like or shingled tier. The usual locations are the posterolateral and occipital scalp regions. The rest of the scalp is often covered with a fine branny scale.

Pathology

The histologic features are those of the underlying disease (neurodermatitis, and occasionally psoriasis) plus extreme degrees of hyperkeratosis.

Etiology

Gougerot stated that this disease is not eczema but an infection of the scalp following impetigo. He claims to have seen the direct transition from impetigo to tinea amiantacea. Furuncles and superficial follicular pustules were present in most of his cases at some stage of the disease. He therefore believed that both the streptococcus of impetigo and the staphylococcus of superficial folliculitis and furunculosis caused the inflammation which results in the original exudation. He stated that in turn the exudation dried out to produce the peculiar asbestos like scales so characteristic of the disease. This view is in direct variance with that of Sabouraud who believed the disease is eczema and begins without any organism being present. Sabouraud further stated that this disease has no connection with the seborrheic or pityriasis groups since he could never demonstrate his seborrheic bacillus in tinea amiantacea. Brown made a very careful study of this disorder and advanced an excellent analysis and adequate explanation for its occurrence. He found that the affection occurred chiefly in adult women who had been subjected to severe nervous and physical strain as a result of problems incurred during the war. Some of his patients were menopausal but this factor did not seem the important one in determining the onset of the outbreak. The majority of them had neurodermatitis generally involving the face, neck and ears sometimes accompanied by recurring attacks of angioneurotic edema. The more typical areas such as the flexures of the arms, axillae, breasts, groin, vulvar and anal areas were frequently although not always involved. When the scalp was involved the clinical picture was that of a scaly dermatitis showing considerable variation in the degree of scaliness: sometimes a mild branny scaliness simulating pityriasis simplex and at other times a more severe seborrheiform dermatitis except that the scales were generally silvery white and rarely greasy. The clinical picture closely simulated that of psoriasis of the scalp—so much that it was difficult to make a differential diagnosis unless there was a confirmatory history or evidence of neurodermatitis elsewhere. In these cases the scales on the scalp became large, pearly or



FIG 228—*Pityriasis amiantacea*. A A very striking patch on the vertex of a patient with lichenoid neuridermatitis of the neck and arms. B *Pityriasis amiantacea* with typical asbestos formation in a patient with psoriasis. This disorder is not uncommonly associated with psoriasis. (Courtesy Dr. W. Herbert Brown and Brit J. Dermat.)

asbestos like ran up the shaft of the hair and when separated formed cylindrical bundles, presenting the typical picture of *tinea amiantacea* or *fausse teigne amiantacée*.

Brown substituted the term *pityriasis amiantacea* instead of the terms *tinea amiantacea* or, as Alibert originally labeled the condition *fausse teigne amiantacée* because it had no relationship whatever to *tinea*. In all, he examined 16 cases—14 in patients suffering from undoubted neurodermatitis and 2 in patients with generalized psoriasis. With regard to sex and age incidence all the patients except one were women, all but two were over 40 years of age.

Brown's observations brought him to the conclusion that neurodermatitis frequently involved the scalp. In this site it is often mistaken for psoriasis or seborrheic dermatitis particularly if the retroauricular folds are also involved and have become eczematized. The clinical picture of *pityriasis amiantacea* (*tinea amiantacea*) appeared most frequently in his experience in neurodermatitis of the scalp. In 14 cases the asbestos scaling was unequivocal and striking in 9. In the other 5 cases it was less prominent but definitely of the same nature. A striking example of it was found in 2 cases of psoriasis. Becker and Obermayer also reported this disease in association with psoriasis of the scalp. *Pityriasis amiantacea* therefore is actually not an etiologic entity. Brown believes that it is simply a phase or stage of reaction of the skin to some underlying disturbance. If this interpretation is correct then it is natural to expect considerable variation in the degree of scaling. Accordingly in some instances the asbestos layer is minimal and in others maximal.

Therapy

The basic therapy of this disease should be directed toward treatment of the underlying neurodermatitis or psoriasis. In addition employment of proper local measures will result in disappearance of the scales and the inflammatory process from the scalp. In the cases reported by Brown, the local application used with most satisfaction was a cream containing 5 per cent of salicylic acid and 5 per cent of oleate of mercury in conjunction with small doses of x-ray therapy. I agree with the effectiveness of this preparation although I do not advise the routine use of x-ray therapy. Grenz ray therapy is permissible if other methods fail. The patient should be instructed to shampoo the scalp with either a tar shampoo or tincture of green soap twice daily followed by the application of a water washable cream. In general the local measures recommended for the treatment of psoriasis of the scalp are effective. The ointment I have found most satisfactory is the following:

Salicylic acid	60
Ammoniated mercury	30
Liquor carbonis detergens	10
Water washable ointment	qs 1000

6 PRURITUS

Clinical Features

While pruritus or itching is a concomitant symptom of a wide variety of dermal diseases it is also the name of a disorder in which itching, smarting, burning, prick

ling, tingling creeping and crawling sensations are the chief symptoms, inciting other lesions but never resulting from them. Hebra was the first medical investigator to distinguish between pruritus the common symptom of skin maladies and pruritus the independent disease. Itching of the scalp where no trace of any local or systemically responsible disturbance can be found is called essential or idiopathic pruritus. Sabouraud characterized it as an intense irritation of the scalp without visible cause.

The onset of the disease whether localized to the scalp or occurring generally over the patient's body is distinguished by one or more of the highly irritating sensations catalogued above. The acute discomfort provokes the patient to rub scratch and otherwise traumatize the affected areas in an attempt to change the pruritic sensation to one of preferable pain. Self control can seldom be mustered even by persons of unusually strong will power. The irresistible urge to scratch becomes pre eminent and brushes combs coarse cloths and even metal instruments have been employed since they simultaneously cover a wider area than that of which the fingernails are capable.

These unhappy efforts of the sufferer to relieve himself result only in temporary alleviation and cause secondary changes such as erythema excoriations with bloody crusts lichenification deep pigmentation and infections from the entrance of bacteria through self inflicted wounds. Some or all of these will be found on examination but, if absent with the skin of the scalp remaining normal the patient's own recitation of his agonizing itching becomes the sole feature of the disorder. The intense irritation shifts from region to region and is felt most acutely at night and in the early morning but less severely during the day. Sleep becomes a thing of the past.

Diagnosis is simplified by the total absence of any cutaneous changes other than those of a traumatic origin. In elderly persons suffering from senile pruritus however where the scalp is only one of the regions affected both atrophy and pigmentation may be evident since senile changes of the skin and tissues are responsible for the itching in these cases. Lymphoblastoma and other diseases must be ruled out by laboratory and clinical examination and in all instances of true pruritus the diagnosis must be fortified by a detailed physical and mental checkup along lines suggested by the etiologic possibilities.

Pathology

Since the very nature of the disease precludes any perceptible structural changes in the affected nerves and peripheral tissues except those which are the direct outcome of trauma or infections due to trauma there has been very little work done on the pathology of pruritus. Bickford however was sufficiently interested to investigate certain aspects of its pathology. He observed that around the original site of the itching sensation there was a sharply defined zone which under the stimulation of rubbing also became a pruritic area. He then incited central itching points by means of histamine pricks in order to study the afferent spread of the margin of the pruritic skin. Thus he discovered was due to a local axonic pathway

not to be confused with hyperaesthesia and vascular flux. Contrary to supposition it is not composed of fibers of the sympathetic nervous system but the mechanism involved is plexiform. Further, Bickford revealed that the itchy skin areas surrounding the original points of maximum irritation can be abolished by asphyxia or cooling which does not interfere with the central pruritic sensation. He associated the nervous mechanism with the sensibilities of prick and tickle but not of touch and believed that it traveled through the anterolateral spinal columns.

An interesting and valuable study was recently made in Tel Aviv by Koenigstein investigating the itch stimuli in animals. He was particularly interested in the sustained type of paroxysm which fluctuates between intensity and exhaustion as in chronic cases of pruritus. He discovered that scratching paroxysms could be induced in dogs by intravenous injections of thalassin in white mice by sensitization with light in cats dogs rabbits and guinea pigs by injections of morphine and other substances into the external cerebellomedullary.

The questions which Koenigstein attempted to answer were threefold. What are the nerve tracts of the itch scratch reflex? How are the pruriginous substances formed and have they properties in common by which they may be collected in a single group? Where in the central nervous system should the site of action of pruriginous substances be sought?

A scratching paroxysm induced by the intravenous injection of thalassin in a dog could be prevented or suppressed by ergotamine tartrate or yohimbine chloride. All other adrenergic or cholinergic substances which stimulate or inhibit the autonomic nervous system failed to affect the itch scratch reflex. In other experiments with dogs larger and smaller sections of the sympathetics were eliminated by unilateral or bilateral operations. The result of these trials supported the conclusion that sympathetic fibers play a part in the conduction of the itch scratch reflex.

Ten experiments demonstrated that ergotamine tartrate and yohimbine chloride exerted a depressant effect on the scratching paroxysms but it was clearly shown that the effectiveness of the drugs was dependent on the site of the action of the pruriginous substances which had been injected. When the injection was in the external there was a central action when it was given intravenously it exerted a peripheral response.

Some of the pruriginous reagents were also found to be stimulants of salivation and vomiting a circumstance which suggested that the responsible centers were similarly situated. The dye, alizarin blue has a strong affinity for tissues and at the same time incites pruritus and produces cramps. When this was injected intracisternally it not only elicited an itching paroxysm but accumulated in a specific location in the lower part of the medulla oblongata bounded cranially by the auditory nuclei and caudally by the corpora testiforme. Koenigstein therefore concluded that the medulla oblongata contains a scratch center.

Etiology

Pruritus has been aptly described as a sensory neurosis. The itching with its subsequent traumatic lesions is clinical evidence that there exists a dysfunction of

the nervous system. The disturbance which provokes this abnormality may have its etiology associated with mechanical factors such as heat, cold and pressure, or an organic functional disease, or an alteration in physiologic activity such as pregnancy and menopause, or an intolerance to certain drugs, such as morphine, antibiotics, etc., or abnormal mental states. Many skin diseases, such as dermatitis venenata and urticaria, also produce varying degrees of itching of the scalp, and these must be carefully eliminated before making a diagnosis of essential pruritus.

The incidence is divided equally among men and women, and while no age level is exempt, there is a tendency for it to be more frequent after forty. Adults with dry and coarse skins suffer from dermatitis hiemalis, or winter prurigo, and sometimes aestivalis, the summer variety, in climates where the temperature is sharply variable. In the same way, a hot bath or cool air or the warmth of bedclothing may precipitate pruritus in persons whom Brenson describes as having a predisposing cutaneous hyperesthesia. Cutaneous irritability resulting from a tight hat band may cause pruritus of the scalp just as the pressure of girdles and trusses with their comparable constriction may cause it elsewhere on the body.

When investigating what seems to be a simple pruritus, examinations must be made to ascertain whether the patient is, without his knowledge, suffering from any of the systemic maladies of which itching is a symptom. Diabetes, Hodgkin's disease or other lymphoblastomas, leukemia, functional or organic disturbance of the intestinal tract (especially parasitic), kidneys, liver, uterus, and ovaries, any one of these may be the essential causative factor. During the climacteric when there is a thorough reorientation of the endocrine glands influencing the nervous system, pruritus is often a concomitant symptom, as it is in pregnancy.

If the most careful dermatologic and medical investigation yields no local or physiologic factors which could account for the intense itching as may be the case the etiologic mechanism is possibly psychosomatic. However, it should always be kept in mind that itching may precede all other symptoms of an underlying systemic disease and it is advisable to search constantly for other manifestations of diabetes, blood dyscrasias, and other causative processes. Also, the search for local causative factors must be a comprehensive and constant one inasmuch as no cure can be expected if some local irritant or allergen remains undiscovered.

Wright pointed out that mental irritations may find their way to the skin either through the phenomenon of conversion involving symbolization or through well known pathways of the autonomic nervous system thereby influencing normal circulation and glandular activity.

Dengrove in his discussion of two patients points out that pruritus may be the chief neurotic symptom arising from the dynamic struggles of the ego with its own special anxiety. His two cases concerned dependent adults with a family history of cutaneous allergy and an arrested emotional development. The onset of pruritus in both of these men was precipitated by the anxiety originating in their forced separation from their emotional support. The first instance occurred because of the death of his all important mother the second because of his removal from his buddies in a closely knit combat crew where there was an extremely inti-

mate personal attachment among the fliers and an important interdependence on one another. Following their removal from the sources of their self assurance the patients suffered from intense pruritus which became the expression of nervous energy through the autonomic nervous system when a psychologic barrier prevented its utterance at the conscious or behavior level. Such a psychosomatic disorder does not express the solution of a conflict but is rather a statement of that insoluble conflict in terms of pruritic sensation.

Wittkower in his study of the psychologic aspects of dermatology believed that by dividing patients into two personality categories: the obsessional and the hysterical one could assign certain cutaneous manifestations to each group. The former includes persons who are uncertain of themselves, who bottle up their emotions and have feelings of inferiority, inadequacy, and general incompetence. They are usually inhibited sexually, shy, quiet, serious, and overconscientious. As obsessional personalities they are prey to fears and doubts and often act compulsively. It is this character type which develops pruritus and its attendant traumatism. Wittkower reminds us that the words dirt and dirty are used both in a literal and a metaphorical sense. Impurity of the skin in popular belief is attributed to dirty habits in the double meaning of the words: certain naive people believe that trespassers of the accepted moral code show their transgressions on their faces. Therefore individuals who feel unclean irrespective of whether they are or not are unhappy within themselves; they often experience itching sensations and an urge to scratch themselves. Basic feelings of uncleanliness are related usually to feelings of guilt.

Scratching itself is an act of aggression. It removes an unpleasant sensation both in reality and phantasy or in a figurative sense. A man scratches his head in perplexity when the task before him seems too difficult and he wishes it were non-existent. However, it remains his problem. His transient sense of frustration arouses his aggressiveness; he needs to repress normally hostile impulses and so deprived of an external outlet by an authority within himself, his aggressiveness may become self-directed and turned toward his own easily accessible skin.

The field of psychosomatic medicine is merely entering its preliminary stages of cultivation. Where dermatology is concerned the association between mental disturbances and some cutaneous manifestations, particularly pruritus, seems clear indeed. There is little doubt that further scientific studies of this interrelationship will be rewarding, although it is also of importance to maintain a balanced perspective with constant searching for other etiologic possibilities.

As a result of his study and treatment of 120 patients with persistent pruritus and excessive excoriation, Seitz formulated certain impressions and hypotheses concerning the psychocutaneous aspects of these disorders. Such patients appear to control and suppress their feelings, presumably because they look upon their own emotional sensitivity as a sign of weakness, inferiority, and vulnerability. They tend to be especially sensitive to affronts and to loss of love and approval to which they react with unexpressed rage. The repressed rage is reacted to in turn with guilt. These unexpressed feelings of rage and guilt, as well as the strong repressed

needs for love, may find symptomatic expression in the form of scratching. Excoriation appears to serve the complex functions of muscular release of the physiologic tension created by repressed rage, atonement for guilt through mutilating self-punishment, and gratification of the need for love through cutaneous erotic masturbatory pleasure. Because of the apparently masturbatory nature of scratching in these patients, excoriation may be associated with feelings of shame, which may account for the clandestine aspects of excessive excoriation. It appears probable that scratching is responsible for the "maddening itching" complained of by these patients, rather than the reverse.

Successful psychotherapeutic management of patients with persistent pruritus and excessive excoriation involves a permissive therapeutic relationship with the patient in which corrective emotional experiences may occur. By the physician's consistent acceptance of him in the face of his expressed inferiority and rage, the patient learns to accept himself and to find constructive rather than personally destructive methods for expressing aggressiveness. If as a result of the permissive therapeutic atmosphere the patient begins to act out his resentments in social settings, an eventuality consistently associated with improvement of the psychocutaneous disorder, limits must be set in order to prevent the development of antisocial behavior. From the psychotherapeutic standpoint, it is especially important that the physician understand that insight alone is not tantamount to cure. Benefit from psychotherapeutic management in such cases derives principally from the corrective emotional experiences associated with the skillfully controlled relationship between the patient and his physician.

Therapy

Since the medulla oblongata seems to be an itch scratch center, drugs which exert a quiescent influence on that portion of the brain are particularly helpful, such as bromides, phenobarbital, and acetylsalicylic acid. Sutton found the latter and amidopyrine most useful. Naturally, the entire physiologic tone of the patient should be improved. The elimination of narcotics, tea, coffee, and alcohol is suggested.

Pruritus of the scalp is often due to some specific agent and responds properly to the removal of said agent and the use of bland, soothing, local measures. On the other hand, idiopathic pruritus of the scalp may require a skillful combination of local and general measures. For the treatment of the acute and extremely pruritic scalp, the initial application of an iced wet dressing may alleviate the patient's discomfort. The solutions of greatest value are weak boric acid and dilute aluminum acetate. These solutions should be applied to the scalp by means of wet packs or towels. Such measures are difficult to use unless the patient is in a recumbent position. It is far more satisfactory to enlist the services of another person or a nurse. The application should be kept extremely cold and administered every two to three hours for approximately twenty to thirty minutes. During this period the use of antihistamines by both the oral and the intramuscular route often results in speedy relief. These drugs are of primary value in the treatment of pruritus due

to the various allergens although the tendency at present is to employ them as a panacea for all the cutaneous ailments to which the flesh is heir. As might be expected they will not perform miracles but they do have a definite and specific value. They are effective primarily in the treatment of acute and to a lesser extent, chronic urticaria of the scalp. They are also of value in various physical allergies of the scalp such as sensitivity to light and in selected instances of local itching. Unfortunately, at the present time the appearance of the symptom of itching on the scalp or elsewhere on the cutaneous surface seems to evoke a conditioned reflex on the part of the doctor. As with Pavlov's dogs, the itch is the ringing bell which stimulates not saliva but the prescription of an antihistaminic cure all. It must be realized that itching is the most common symptom encountered in dermatologic practice and may be observed in any one of several hundred diseases.

The antihistaminics are a group of substances which in principle of action resemble the antibiotics. In the allergic conditions in which they are used theory has it that histamine is produced and acts upon the cells that are sensitive to it. Administration of the antihistaminics interferes by biologic competition with this action of histamine thus making its effects null and void. Today with many effective antihistaminics in popular use there is still room for improvement in the production of such compounds having even greater effective properties and still lesser toxic and adverse side effects. With the antihistaminics the dosage schedule to produce and maintain the desired improvement must be ascertained in each case. The usual range for adults is from 12 to 400 or even 600 mg. in twenty four hours given in divided doses of 4 to 100 mg. each. The drugs are rapidly absorbed from the gastrointestinal tract and rapidly fixed, destroyed or excreted. Experiments indicate that in man effective blood levels are reached within the first thirty to fifty minutes after ingestion on an empty stomach and are maintained for one to two hours thereafter. Administration of the drugs must therefore be so spaced that the effective levels are achieved at the times when the expected manifestations would be most disturbing. The side effects encountered with Pyribenzamine, Benadryl and other antihistaminic agents are similar but not always identical. In the same person one drug may be without significant side effects whereas the other produces disagreeable sequelae or one drug may produce side reactions different from those produced by the other. It is therefore wise to switch to a trial of some other member of the series when the first is not well tolerated. In cases in which such side effects as drowsiness or sedation are marked but in which continuation of these drugs is urgently required attempts can be made to counteract the drowsiness by the addition of such drugs as Benzedrine or Dexedrine sulfate or ephedrine when overstimulation, jitteriness or sleeplessness occur the addition of barbiturates or of other sedatives may be added. It must always be kept in mind that any of these drugs may initiate, prolong or aggravate the pruritic state.

There have also been numerous reports stating that the antihistamine drugs in ointment form were of value in the treatment of many itchy skin diseases. Although this is true they are also externally irritating in many of these conditions and may aggravate the original disease. Antihistaminic ointments should be employed with caution on a pruritic scalp especially when a dermatitis is present.

The following preparations are often employed in the treatment of nonspecific pruritus of the scalp (see Formulary, page 551, for additional local preparations)

Ointments

Menthol liquef	0 2
Phenol liquef	1 0
Hydrophilic ointment U S P	q s 100 0
Liquor aluminum subacetate	5 0
Anhydrous lanolin	10 0
Cold cream, unscented	15 0
Eurax ointment	q s
Antihistamine powder	1 0 5 0
Water washable ointment (Many such preparations are commercially available)	q s 100 0
Menthol liquef	0 2
Benzocaine	3 0
Cold cream, unscented	q s 100 0

Lotions

Menthol liquef	0 2
Liquor carbonis detergens	5 0
Water	q s 100 0
Benzocaine	1 0
Chloral hydrate	4 0
Castor oil	5 0
Alcohol	20 0
Water	q s 100 0
Glycerin	5 0
Phenol	1 0
Camphor	3 0
Water	q s 100 0
Menthol liquef	0 1
Boric acid	3 0
Water	q s 100 0

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CHAPTER VII

SCALP INVOLVEMENT FROM SKIN DISEASES

INTRODUCTION

Many disorders of the skin frequently involve the scalp as well. In the majority of such instances, the scalp lesions are merely representative of the typical lesions present elsewhere on the body surface. However, variations in the characteristic appearance of such lesions are often encountered on the scalp due to the different morphologic and physiologic components of its surface and structure.

In order to simplify the difficulties of reference to the various skin disorders and diseases, they are presented in alphabetical order. If the disease was of only slight import as far as the scalp was concerned, a brief description and discussion sufficed. The more important skin diseases affecting the scalp are described in detail with respect to their diagnosis and therapy in that anatomic location.

1. ACNE NECROTICA MILIARIS

Clinical Features

Persistent itching of the scalp causes the patient to consult his physician for relief. When examined meticulously, the adult scalp exhibits a few minute, yellow pustules situated at the ostia of the hair follicles. These pustules are of pinhead size sometimes with a conical crust superimposed on a moist base. Usually only three or four of these tiny, oozing points confined to a small area or widely scattered may be found on an entire scalp but in more severe cases the brow and areas of the cheek, neck and chest may be simultaneously involved.

On the scalps of infants and young children the features are somewhat different. Onset is preceded by a generalized erythema. Then follows a widely distributed, milary, follicular pustular eruption. Crusts form and fall, but the entire process may continue for months with attacks following one another in close succession.

Montgomery reported 25 adult cases with similar development. Pruritus preceded the appearance of minute vesicles which ruptured in six hours, first oozing serum and then crusting. Of this series only a few ulcerated. While some say that chronic cases eventually develop into true acne necrotica Savill could not substantiate this. One of her cases recurred at intervals over a period of twenty years and never developed into the more serious disease.

There may be some difficulty in distinguishing acne necrotica miliaris from Bockhart's impetigo which is also an inflammatory pustular eruption at the site

of the pilosebaceous orifice. The yellow pustule is usually centered by a hair, its shape is hemispherical rather than conical, and the size varies between pin point and lentil instead of being uniformly minute as in *acne necrotica miliaris*.

Pathology

The lesion is a simple intradermal vesicle filled with serum. Staphylococci are the major etiologic organisms. The histologic picture is similar to that of a small or early lesion of *acne varioliformis*.

Etiology

Sabouraud considered this disease merely an abortive form of *acne necrotica*. By culturing the crusts, he found *Staphylococcus aureus* and other microorganisms. Montgomery and Ormsby point out that the characteristic lesions frequently follow other scalp disorders such as suppurating wounds, furunculosis, and contact dermatitis. The incidence of *acne necrotica miliaris* is usually higher after middle age. Men are more prone to it than women in the ratio of two to one.

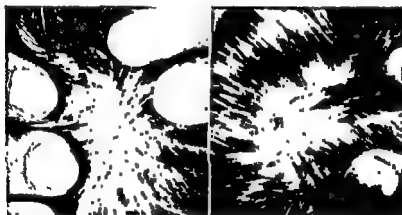


Fig. 229—*Acne necrotica miliaris* showing perifollicular vesicopustules of an intensely pruritic nature. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph by Dr. Hamilton Montgomery.)

Therapy

In general, the measures advised under the treatment of follicular impetigo and *acne varioliformis* are adequate (pages 248 and 411). Improvement but not a complete cure may follow the use of any mild mercurial, tar, or sulfur application, whether in ointment or lotion form. Local therapy should be combined with systemic measures (as described on page 252).

2. ACNE URTICATA

Acne urticata is a somewhat vague entity described by Kaposi, Touton, Lowenbach, and others as a condition in which urticarial wheals and papules appear on the scalp, face, and other portions of the body of neurotic individuals. The primary lesion, often preceded by itching and burning, is a small wheal which gradually enlarges in size. In a short time, the center of the wheal becomes paler, flatter,

and vesicular, with eventual crust formation. The crust falls, leaving a small scar in the scalp or on the face which in time becomes depressed and of a shining white color. As Adamson suggested, the wheals and papules are probably secondary to self inflicted traumatic irritation. Lesions of this type are not at all uncommon in the scalp but are actually neurotic excoriations and are more completely discussed in the chapter on neurodermatoses.

3. ACNE VARIOLIFORMIS

Clinical Features

This papular eruption has the distribution of a pustular seborrheic dermatitis and the morphology of a papulonecrotic tuberculid. The vertex, the temporal regions, the anterior portion of the scalp near the hair border, and the adjacent forehead may become the sites of acne varioliformis. The lesions are discrete, indolent, and extremely pruritic papules which develop into reddish brown, crusted vesicopustules with an umbilicated center. Montgomery notes that in some cases the peripheral extension of the papulopustules may create a narrow, annular, seropurulent chamber with a depressed, firm center.

The papules form about the pilosebaceous orifice and therefore are usually pierced by hairs. These hairs are shed with the crusts about a week after the appearance of the papules, at the time when the inflammation has begun to subside. The shedding of the crust leaves a superficial ulcer which heals as a permanent, slightly depressed, circinate scar due to necrosis of the follicle and the perifollicular tissue. When these scars are closely aggregated, they cause a patchy, cicatricial alopecia resembling the pitted scars of smallpox. Similar lesions may be present on the face and chest.

Because of the extreme pruritus and burning sensations present in the early stages of acne necrotica, constant scratching is likely to modify the appearance of the lesions so that they will not always appear as described, but will have modifications and complications caused by trauma. Although the life of each papulopustule is comparatively brief, others constantly continue to appear, thereby producing a chronic disorder which undergoes periodic remissions and exacerbations over a period of years.

Pathology and Etiology

Sabouraud believed that his seborrheic microbacillus was always present first, and superimposed on that came a staphylococcus as an active agent, infiltrating the upper part of the hair follicle and then spreading to include the entire follicular apparatus including the sebaceous gland. Several investigators have cultured both streptococci and necrotizing staphylococci from the lesions. Intradermic injections of these microorganisms supposedly resulted in the formation of lesions identical to those of acne varioliformis.

McCarthy, examining the crusts, discovered that they contained clumps of staphylococci at various levels in the marginal zone with a mass of microbacilli centered in the area corresponding to the follicular orifice. Necrotic tissue and inflammatory cells replaced the pilosebaceous structure and follicular epithelium. In some instances the latter had completely dissolved. The skeletal remains of the

sebaceous glands can at times be located but more often they have disappeared without trace. Blood vessels adjacent to the necrotic areas may be thrombosed, and the necrosis may have attacked not only the follicular apparatus but the collagen and elastic tissues as well.



Fig. 230 —Acne varioliformis, showing typical lesions in a common location. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph by Dr. I. Pels.)



Fig. 231 —Acne varioliformis showing typical lesions and atrophic, rounded scars on cheeks and nose. (Courtesy Dr. Frank Combes.)

In some active lesions the follicles are surrounded by a dense, round cell infiltration with epithelioid cells and tubercloid structure. Whether these cases are actually acne varioliformis or tuberculids is problematic. A survey of the litera-

ture would indicate that an inactive or burned out tuberculous process may be the contributory factor in some of these cases which were incorrectly labeled acne varioliformis

Acne varioliformis flourishes on an underlying seborrheic "soil." The disease attacks obese, inactive men and women in the middle period of their lives. However, it also occurs among young debilitated individuals. It is also important to rule out the possibility of a septic focus in the teeth, tonsils, sinuses and, in women, the cervix. These underlying systemic factors may pave the way for a superimposed, specific microbial infection.

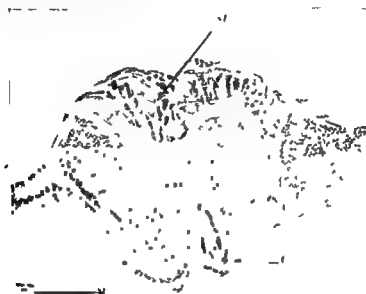


Fig 232—Acne varioliformis. Above, at *st* is a pustule containing large numbers of staphylococci. Below at *f*, is the follicle containing the primary culture of the seborrheic bacillus. (Courtesy McCarthy. Diseases of the Hair. Reproduced from Sabouraud. Affections du Cuir Chevelu, Masson & Cie.)

Therapy

The first step in the treatment of this disease is the correction of underlying systemic dysfunction. In the usual obese patient, the diet should be similar to that employed for the correction of other recurrent pustular conditions. In general a high protein, low fat and low carbohydrate regime is advisable. In addition the following foods should be interdicted: chocolate, nuts, shellfish, highly seasoned and greasy foods, iodides and bromides in any form, and alcohol. The occasional patient may also be benefited by immunotherapy (staphylococcus toxoid, autogenous vaccines).

Local measures are initiated with daily scalp shampoos. In the usual instance this may be followed by the application of antibiotic and antiseptic lotions or ointments containing bacitracin, furazolidone, ammoniated mercury, salicylic acid, a member of the quinoline family, hexachlorophene or bichloride of mercury. Fre-

quent exposures to ultraviolet rays are of value. For this purpose, a small cold quartz type of applicator is useful. Weekly applications of powdered carbon dioxide in acetone are of value in resistant and recurrent cases. The disease has a definite tendency toward recurrence, and therapy must be continued for long periods of time after complete subsidence of the active lesions. Relapses are also not infrequently observed following neglect of the general measures advised in the preceding paragraph. The following prescriptions are often therapeutically effective.

(1) Quinolor Compound Ointment	20.0
Ppt. sulfur	2.0
Castor oil	5.0
Petrolatum	qs 100.0
(2) Ammoniated mercury	5.0
Salicylic acid	1.0
Cold cream U.S.P.	qs 100.0
(3) Diodoquin or Vioform	2.0
Bacitracin ointment	qs 100.0
(4) Hexachlorophene	1.0
Bichloride of mercury	0.06
Salicylic acid	3.0
Resorcin monoacetate	3.0
Spirits of lavender	15.0
Castor oil	5.0
Alcohol (70 per cent)	qs 100.0
(5) Bacitracin, penicillin, terramycin, or aureomycin ointment	
(6) Quinolor Compound Ointment	5.0
Bacitracin ointment	qs 30.0

4 ACRODERMATITIS CONTINUA (HALLOPEAU)

Involvement of the scalp occasionally occurs in this disease. The lesions appear as scaling, crusted patches which can be scraped off to reveal an underlying purulent and exudative surface.

5 ALOPECIA (NONSPECIFIC)

Alopecia of a nonspecific and noncicatricial type and either partial or complete in extent may occur as follows:

1 In association with various skin diseases such as acanthosis nigricans, dermatomyositis, erysipelas, exfoliative dermatitis, ichthyosis, and hypomelanotic reticulosis.

2 In association with the exanthems, various toxic states and high fever, a diffuse shedding of scalp hair may occur. In addition to the exanthems, this phenomenon occurs in association with other diseases such as grippe, influenza, pneumonia, ratbite fever, septicemia, and typhoid fever.



Fig 233—Temporary alopecia occurring seven weeks after erysipelas. Complete regrowth of normally pigmented hair four months later. (Courtesy McCarthy ■ seasons of the Hair)



Fig 234—Extensive loss of hair following influenza. (Courtesy O'Donoghue and The Hair)

3 In association with various other temporary or prolonged disturbances and diseases such as cachexia from any cause diabetes mellitus disseminated lupus erythematosus endocrine dysfunction lipid proteinosis tuberculosis Vogt-Koyanagi syndrome surgical procedures neuroses and pregnancy

6 ATOPIC DERMATITIS (DISSEMINATED NEURODERMATITIS)

This disorder may be classified as a chronic but superficial skin disorder characterized by pruritus thickening and lichenification of the skin. Over a period of years poorly defined plaques appear on the face neck and flexural aspects of



Fig. 235.—Atopic eczema in stage of extensive exfoliation with scaling of face and scalp

the elbows and knees. The disease often begins in infancy and usually occurs in individuals with a high familial incidence of allergic disease. Although the disturbance is basically allergic in origin there is no question of the fact that psychosomatic and other factors are frequent predisposing and exacerbating mechanisms.

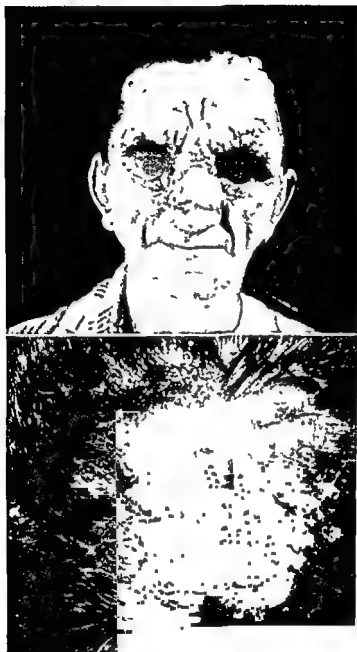
In those cases where specific allergens are responsible for the dermatitis, their elimination may result in cure. Although this disease may involve the scalp, it is not one of the more frequent sites. This disease is also allied to the localized form of neurodermatitis and, as such, it is discussed elsewhere (page 375). From a therapeutic standpoint, both systemic and local measures are of importance. Although desensitization procedures may be of value (when sensitivity has been demonstrated to a contactant, inhalant, or ingestant), elimination of the suspected agent or avoidance of exposure to it are of greater value. This disease is one of the major dermatologic problems and a discussion of it is out of place in this volume. Suitable descriptions may be found in any of the standard textbooks on dermatology. The local manifestations of its involvement of the scalp are comparable to those described under the title of Circumscribed Neurodermatitis and a discussion of causative mechanisms, pathology, and therapy are described under that heading (page 375).

7. DARIER'S DISEASE

Darier's disease, or keratosis follicularis, displays a wide variety of lesions, depending on their location. The process as it involves the scalp tends to have a seborrheic distribution with a special predilection for sites of intense perspiration.



Fig. 236—Darier's disease. Typical lesions as they involve the scalp. The temporofrontal regions are most commonly affected. (Courtesy McCarthy, *Diseases of the Hair*.)



B

Fig 237 —Darier's disease *A* Vegetative type with involvement of the face and scalp margins
B Vegetative lesion in the scalp (Courtesy Dr Herman Beerman and Arch. Dermat. & Syph.)



Fig 238 —Darier's disease associated with almost total alopecia (Courtesy Dr D Welton and Arch Dermat & Syph)



Fig 239 —Darier's disease. Photomicrograph of skin biopsy showing dyskeratotic cells (Courtesy Dr D Welton and Arch Dermat & Syph)

In addition to the hairy portions of the scalp it may be situated on the temples, the face, particularly in the nasolabial folds the retroauricular regions neck and axillae the presternal interscapular, umbilical and genitocrural regions and the mons veneris back gluteal regions dorsa of the feet soles hands and palms

The lesions which are often bilateral and symmetrical are reddish yellow and may be plaque-like fungating condylomatous papillomatous, cockscomb-like, or tumorous. Umbilicated papules no larger than 1 cm. are fairly common and morphologically resemble those of molluscum contagiosum.

Essentially the initial papules are the result of a keratosis of the follicular orifices pinhead in size and of the same color as the adjacent integument. As the disease progresses the papules become red brown in color, covered with a gray or brown firmly adherent crust, and greasy to the touch. At this stage the lesions resemble the crusting forms of seborrheic dermatitis.

In time these papules coalesce until the whole area is occupied by a mass of small dark brown tumors and hyperkeratotic growths involving the interfollicular tissues as well as the follicles and rendering them hypertrophic. Superficial ulcers may form at the sites of the follicular orifices and a mucopurulent malodorous fluid is secreted by the vegetating mass. Secondary staphylococcal infection may occur with its attendant pus formation. While hair loss is not immediate, it may follow as the disease progresses. Cases which are noted in infancy are usually on an hereditary basis and often of a more severe nature. Welton even reporting a case of alopecia totalis with the denuded scalp presenting symptoms characteristic of Darier's disease.

Histologically the process is an epithelial one. The chief features are dyskeratoses the corps ronds of Darier and grain shaped formations. The microscope also reveals hyperkeratosis and parakeratosis acanthosis lacunae in the epidermis and a pronounced basal cell hyperplasia. The uneven surface of the lesion contains invaginations filled with horny plugs while the surrounding epithelium shows the atypical proliferation of the basal cell layer.

When the vegetative type attacks the scalp the epithelium surrounding the hair follicle sends out processes resembling epithelioma with lumen in their depths. The cells of these tubelike projections are cuboidal and epithelial. Beerman points out that it is these processes which account for the luxuriant papillary growth.

The consensus now is that Darier's is not a follicular disease at all but affects the hair follicles only incidentally. Hidaka found that 68.6 per cent of the lesions in his cases were nonfollicular that 30.7 per cent were situated near follicles but only one was actually perifollicular.

Differential diagnosis may be necessary where familial benign chronic pemphigus is concerned since the latter has lacunae slight basal cell proliferation and extensive rather than slight acantholysis. The complete lack of hyper- or parakeratosis indicates to some observers that Darier's disease is not in the picture at all. Nevus syringocystadenomatosus papilliferus of the scalp may complicate the diagnosis where the vegetating type is concerned. The most notable clinical difference

the single lesion or small group of lesions presented by the nevus rather than the more diffuse involvement of Darier's disease. Histologically the discrepancy is even greater. Nevus syringocystadenomatosus papilliferus like familial benign chronic pemphigus, lacks any trace of hyper-, para-, or dyskeratosis lacunae, and basal cell proliferation. It is primarily a lesion of the sweat apparatus showing regularly intracystic papilliferous projections covered by two layers of cells columnar on the outside, cuboidal within.

Local measures are of little value in Darier's disease. The ingestion of large doses of vitamin A is an aid in control of the more extensive forms of this disease.

B DERMATITIS HERPETIFORMIS

This disease rarely involves the scalp alone although an occasional vesicopapular lesion or group of herpetic lesions may be observed on the scalp during the course of the disorder. When the bullae dry the scalp is left covered with large, flaky scales.



Fig. 240—Dermatitis herpetiformis of the scalp. (Courtesy Dr. Frank Combes.)

9 DERMATITIS MILDICAMINTOSA

Under this title, the manifestations on the scalp induced by the internal administration of drugs will be considered. This includes those ingested, absorbed through the mucosa, and those parenterally administered.

Arsenic. Inorganic arsenic may be an uncommon cause of keratoses and epithelioma of the forehead and scalp. In several cases of alopecia areata arsenic and lead were present in increased amounts in the body fluids and tissues. The presence of these heavy metals in the body was supposedly accounted for by the wide spread prevalence of arsenic and lead in unwashed fruits, vegetables, and other foods due to spray residues, insecticides, and various other forms of contamination.



Fig. 241.—Alopecia following drug ingestion (arsenic). (Courtesy Dr. Frank Combes.)

Atabrine. The lichen planus like eruptions and exfoliative dermatitis attributed to Atabrine are occasionally accompanied by a nonspecific diffuse alopecia.

On the basis of histologic examination Schamberg stated that persistent alopecia following Atabrine dermatitis is the result of destruction of the hair follicles by a deep inflammatory process sufficiently severe to destroy the hair follicles and the sweat ducts. Similarly cutaneous atrophy occurs when Atabrine dermatitis destroys the entire thickness of the epidermis. Temporary alopecia and anidrosis and absence of atrophy in skin areas previously the site of dermatitis may be presumed to result from a less acute and less prolonged inflammation, from which the hair follicles, sweat apparatus and epidermis are able to recover.

Barbiturates Exfoliative dermatitis due to this drug produces redness scaling and diffuse alopecia of the scalp. Similar changes in the scalp may occur during the course of an exfoliative dermatitis produced by arsphenamine carbarsone dinitrophenol gold mercury penicillin streptomycin and sulfonamides. If the patient recovers from the disease the hair grows in again although it is usually quantitatively less.

Bromides and Iodides In acneiform and granulomatous eruptions encountered following iodide or bromide therapy, a few papulopustules or even a fungoid nodule may be encountered on the scalp.

Cytarabine hydrochloride The use of this drug was followed by a diffuse and extensive alopecia.



Fig. 249.—Lichen planus-like eruption of face and scalp margins following drug ingestion (quinacrine). (Courtesy Sutton and Sutton: Handbook of Diseases of the Skin.)

Gold Alopecia areata and totalis have occurred following gold therapy.

Propylthiouracil Diffuse hair loss has been reported following use of this drug in the treatment of hyperthyroidism.

Quinine hydrochloride Alopecia areata has occurred following the intravenous use of this drug.

Thallium The only internally acting drugs known to produce alopecia with any degree of consistency are the thallium salts. Unfortunately, this effect is often associated with severe manifestations of systemic toxicity and even death. In a group of children with ringworm of the scalp who were treated with this drug, a miscalculation in the dosage resulted in the death of several of them. In view of this possibility and the high incidence of toxic reactions from this drug, it is there-



Fig 243 Alopecia following drug intoxication (cybernetic hydrochloride)



Fig 244 Alopecia of a permanent nature following use of a depilatory containing thallium acetate (Courtesy McCarthy Diseases of the Skin)

apeutically contraindicated. It would be of great value to have a drug that could consistently produce temporary alopecia of the scalp without untoward reactions elsewhere, but thallium is not the answer.

Experimental work by Uruena, Pardo Castello, Andrews, and others showed that the most satisfactory dose of thallium acetate was 8 mg. per kilogram of body weight, administered as a single dose. The hair fall begins on the seventh day and is usually complete on the fifteenth to eighteenth day. A small fringe of hair is usually left on the anterior frontal and lateral scalp margins. Regrowth occurs in three months although a downy fuzz appears one month after the ingestion of the drug.

Smaller amounts are ineffectual and larger amounts produce salivation, myositis, neuritis, lethargy, and headache. With overdosage, severe reactions occur in the gastrointestinal tract and central nervous system with possible fatal termination. Surprisingly enough, this drug is even more toxic to adults than to children, possibly due to the basic effects of this drug on the endocrine glands and the autonomic nervous system. The effects of this drug are generally considered due to a temporary arrest of the action of the endocrine glands and the autonomic nervous system on hair growth. It has also been postulated that thallium may react with the free sulphhydryl groups of cysteine and homocysteine, resulting in cystine deficiency and thus inhibiting normal hair growth. Fleisch and Goldstone recently proved, however, that the depilatory effect of thallium is not due to a direct interference with normal sulphhydryl metabolism although it may interfere with the synthesis or utilization of cystine.

In former years, a depilatory cream (Koremlu) containing thallium acetate, was marketed for local application as a hair remover. Needless to say, it not only was inadequate as a depilatory, but was responsible for a number of toxic and dangerous reactions.

10 DERMATITIS VEGETANS

This local infection involves the scalp, axillae, genitalia, groin, and mucocutaneous junctions. On the scalp, it is characterized by military papulopustules appearing in groups with subsequent coalescence and the formation of crusted, elevated, vegetative patches clinically resembling blastomycosis. It responds readily to local antiseptic measures.

11 ERYTHREDEMA

Children with this avitaminotic disorder present a picture of abject wretchedness. In fact they are so miserable that they literally tear out their hair. Accordingly erythredema may be classified as a causal agent of trichotillomania.

12 EPIDEMIC EXFOLIATIVE DERMATITIS (SAVILL)

An epidemic of this disease occurred approximately fifty years ago and a few cases were subsequently reported. It is questionable as to whether such an entity exists although the disease was carefully studied at the time of its original descrip-

tion. It occurred in two distinct clinical types, one resembling a moist and extensive eczematous dermatitis, the other being a dry exfoliative form. The regions most frequently involved were the scalp, face and upper extremities. The basic skin lesions were grouped acuminate papules around the follicular orifices. In severe instances the hair and nails were lost.

The etiologic agent was not specifically detected although a member of the staphylococcus family was suspected. Treatment in general did not appear to influence the course of the disease. Although the majority of patients recovered there was a mortality rate of approximately 10 per cent. Inasmuch as this disease has not been observed in recent years it is quite possible that it is merely a variant of some entity now classified under a more specific heading.



Fig. 245.—Dermatitis vegetans. (Courtesy Dr. F. Reiss.)

13 EPIDERMOLYSIS BULLOSA

Epidermolysis bullosa of the dystrophic type is not infrequently associated with changes involving the scalp. In this rare hereditary disease the hair is affected along with the changes in other ectodermal organs. Wende described the occurrence of complete and permanent alopecia in two infants from the same family. These children were born with thick hair which fell out at the usual time and never grew back. They also showed the usual features characteristic of this bullous dermatosis. Several other variations of alopecia in association with this disease have been described and are included elsewhere under the classification of congenital alopecia.



Fig 246 —Epidermolysis bullosa hereditaria with arrangement of bullae in concentric lesions Alopecia of scalp as well as involvement of eyebrows eyelashes, and hands was noted (Courtesy Sutton and Sutton Diseases of the Skin Original photograph by Dr ■ W Wende)



Fig 247 —Erythema multiforme (erythema annulare centrifugum) of the scalp margins (Courtesy Dr Frank Combes

14. ERYTHEMA MULTIFORME

In the various forms of this disorder, including the more severe manifestations grouped under the title Stevens Johnson disease, there is little or no involvement of the scalp. An occasional bullous or papular lesion or group of lesions may be seen on the scalp during the course of this disease.

15. ERYTHEMA NODOSUM

This symptom complex has been observed as a rare complication of an active fungus infection of the scalp.

16. ERYTHRODERMA

Erythroderma or exfoliative dermatitis is a generalized, scaling eruption involving the entire body and usually showing an associated scalp, red scalp with an alopecia of varying extent. Some of the causes of this extensive skin disorder with scalp changes of scaling, redness, and alopecia include:

a. Drugs internal: antipyrine, arsphenamine, Atrazine barbiturates, carbarzone, dinitrophenol, gold, mercury, penicillin, quinine, serum, streptomycin, sulfonamides.

b. Drugs external: acriflavin, arnica, chrysarobin, picric acid, mercury, resorcin, salicylic acid, sulfur.

c. Lymphoblastomas (leukemia, Hodgkin's disease, granuloma fungoides and follicular lymphadenopathy).

d. Skin diseases: atopic eczema, dermatitis venenata, eczema, infectious eczematoid dermatitis, lichen planus, pityriasis rubra pilaris, psoriasis, and seborrheic dermatitis.

17. ERYTHRODERMA DESQUAMATIVA (LEINER)

In this exfoliative dermatitis of breast fed infants, the scalp is covered by a severe and extensive seborrheiform eruption as manifested by yellow crusts and scales on an inflamed, reddened skin. Leiner differentiates this affection from impetigo or dermatitis exfoliativa neonatorum (Ritter), which it may closely resemble by its chronicity and the severe seborrheic type of scalp involvement.

18. EXANTHEMS

(Scarlet Fever, Measles, Rubella, Variola, and Varicella)

In exanthems, it is not unusual for the scalp to share in the process and show lesions typical of the stage of the disease elsewhere on the body. Vesicular, pustular, and ulcerative lesions of variola and varicella in the scalp may leave sequelae in the form of small areas of cicatricial alopecia. In addition a diffuse shedding of scalp hair may occur during the course of these diseases or during the period of convalescence.

19 ICHTHYOSIS

The hair is dull lusterless and dry, and the scalp is covered with a fine, dry scale in mild forms of this disease. In the more severe examples of this malady the vertex is covered with thick gray, quadrangular rough edged scales adherent centrally to the scalp (crocodile scales)



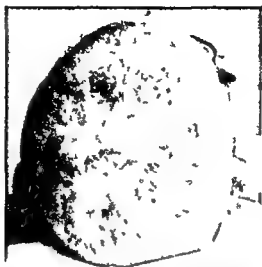
A



B

Fig 248—Ichthyosis. A Harlequin fetus showing involvement of scalp. B Extreme degree of ichthyosis with alopecia. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph of harlequin fetus by Dr C. M. G. Wakeley.)

Ichthyosiform Erythroderma (erythrodermie congenitale ichthyosiforme) — The scalp is dry and scaly in this unusual disease but the hair is not necessarily affected



B

Fig. 249—Keratosis blepharorrhoeica. *A* Scaly crusted lesions and alopecia (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photograph by Dr. H. L. Keim.)
B Corona gonorrhoeica (Courtesy Dr. F. Combes.)

Ichthyosis Follicularis—This peculiar disease is characterized by an ichthyotic skin keratosis pilaris like papules, diffuse alopecia of the scalp eyebrows and eye lashes, trachoma and conjunctivitis

20 INCONTINENTIA PIGMENTI

In this rare pigmentary dermatosis spotty alopecia has been reported In one instance the scalp showed numerous atrophic sharply circumscribed bald spots and brown areas of pigmentation In another instance the scalp showed scleroderma like changes and alopecia clinically similar to the circumscribed form of scleroderma referred to as *en coup de sabre*

21 KERATOSIS BLENNORRHAGICA

This chronic inflammatory dermatosis is due to the gonococcus and occurs in conjunction with gonorrheal infection of the genital tract and joints It is characterized by a symmetric eruption of horny conical nodules pustules and crusts on the palms and soles The scalp and forehead may show similar lesions



Fig 250—Keratos s blennorrhagica with scaling crusted lesions and corona gonorrhoeica (Courtesy Clay Adams Co)

22 KERATOSIS PILARIS

Clinical Features

This disease primarily involves the extensor aspects of the arms and thighs as discrete goose flesh papules imparting a nutmeg grater feeling to the skin The papules are situated at the mouths of the hair follicles and are composed of sebum and epithelial scales These papules are usually of pinhead size grayish in color and, although often pierced by a hair the latter is more often curled up inside.

Over a long period of time the pilosebaceous apparatus may be destroyed and replaced by minute white patches of cicatricial alopecia. In rare instances the disease affects the outer third of the eyebrows and the scalp and the resultant cicatricial alopecia resembles that of folliculitis decalvans and pseudopelade. The diagnosis may be simplified by the finding of follicular plugs at the margins of the cicatricial patches. It is questionable whether such cases are a rare form of keratosis pilaris or actually follicular types of lichen planus.

Ulerythema ophryogenes as first described by Trenzer and Unna clinically presents the same lesions as keratosis pilaris. Supposedly the typical horny papules first appear on the eyebrows and spread to the scalp and extremities. The disorder may persist for years without change or through the process of superficial inflammation and atrophy progress to final small white patches of cicatricial atrophy. Dubreuilh, MacLeod, Jackson and Brocq considered it a form of keratosis pilaris which it is. The former term should be dropped.



Fig. 251.—Keratosis pilaris. H, Hair shaft; M, muscle; A, acute bend in follicle; G, coiled gland. (Courtesy Sutton and Sutton, *Diseases of the Skin*. After Unna.)

Pathology

The papules are produced by a hyperkeratosis about the follicular orifice filling them with horny plugs in which are coiled one or more fine hairs. The surrounding epidermis is also hyperkeratotic. There may be a slight inflammatory reaction in the surrounding corium. A moderate to severe degree of atrophy of the follicle (including the hair papilla), sebaceous glands, and erector pili muscles is evident.

Etiology

Keratosis pilaris is probably a congenital disease related to ichthyosis, subnormal sebaceous glands, and increased cutaneous requirements for vitamin A.

Therapy

Frequent bathing and the use of strong soaps have been advised. I prefer mild abrasives and local exfoliative procedures including ultraviolet radiation fol-

lowed by the use of bland emollients. The following emulsion has proved very helpful.

Acid salicylic	30
Sodium lauryl sulfate	10
Cetyl alcohol	100
Glycerin	100
Water	qs 1000

This preparation should be gently massaged into the affected area several times daily.

General measures should include the avoidance of wool, mild exercise, and the ingestion of 150,000 to 300,000 units of vitamin A daily. The patients should be observed at intervals in view of the possibility of hypervitaminosis A.

23. LICHEN PLANUS

Clinical Features

Lichen planus is a disease which rarely affects the scalp. This has been a source of great confusion to students of the disease because of the appearance in the literature of many reports of various unusual scalp entities associated with lichen planus of the body. This confusion may be dispelled if it is realized that although such cases are associated with lichen planus, they are actually a special entity and are described as such under the heading of follicular lichen planus or lichen pilaris seu spinulosus. Accordingly, the elimination of this group leaves only a very small number of cases of lichen planus in which typical lesions of the disease may be observed on the scalp. In such instances, a few or several small flat topped purplish red papules are found scattered among the hairs. These lesions are similar to those present elsewhere on the body and present no difficulty in diagnosis. The papules may group together to form a confluent patch or plaque, most often in the vicinity of the nuchal region. In such instances, the differential diagnosis from patches of circumscribed neurodermatitis may be difficult, although the presence of typical lesions elsewhere and histologic examination will eliminate the source of confusion.

Pathology

Laymon reported that lichen planus of the scalp usually presents histologic features which are identical with those characterizing lichen planus on the other parts of the body. Relative and absolute hyperkeratosis, increase in the stratum granulosum, plate-like acanthosis and liquefaction degeneration of the basal cells constitute the important changes which affect the epidermis. Parakeratosis was not noted in his cases. He stated that lesions involving the scalp, as in other areas of the body show a sharply margined infiltrate in the corium composed chiefly of lymphocytes with occasional polymorphonuclear leucocytes. This infiltrate is not invariably present however, as was exemplified in one of his cases in which there were characteristic papules of lichen planus on the scalp. Histologic examination showed only a spotty infiltrate in the upper portions of the cutis. He also noted that in several cases there was a sharp band of infiltrate hugging the follicle as well as a diffuse sharply margined infiltrate between the follicles.

Etiology

The cause of lichen planus is unknown. It is generally assumed to be related to systemic disease and possibly to either physical or emotional strain.

Therapy

No specific treatment is indicated for lichen planus of the scalp. In general, the measures to be employed are similar to those used in the treatment of lichen planus elsewhere on the body surface. Internal measures include the use of heavy metals such as bismuth mercury, or arsenic. The scalp lesions rarely require local applications, although in instances of severe pruritus, the application of a 50 per cent trichloroacetic acid solution directly to the lesions will often result in improvement. This application requires neutralization by sodium bicarbonate solution after a two minute period. This improvement, of course, is preceded by a short period of irritation at the site of application.

24 FOLLICULAR LICHEN PLANUS (LICHEN PILARIS SEU SPINULOSUS)

Clinical Features

The childhood disease entitled lichen pilaris seu spinulosus is characterized by the sudden appearance of groups of papules on the body and proximal aspects of the extremities. The papules are pinhead in size, follicular in location, at first red then normal in color and surmounted by a horny spine. This filiform spine, considered characteristic, projects one sixteenth of an inch above the surface and imparts a nutmeg grater feeling to the skin. Removal of this spine leaves a depression in the papule, unlike *keratosis pilaris* in which removal of the horny plug removes the entire lesion. Other differential factors which typify *keratosis pilaris* include the slow evolution and discrete ungrouped nature of its papules, and the absence of redness at any stage. Lichen pilaris seu spinulosus is considered by some dermatologists as either an avitaminosis or a variant of lichen planus.

A source of confusion for many years has been a group of cases with lichen spinulosus of the extremities and trunk in which this disease was preceded by a scalp eruption supposedly resembling folliculitis decalvans. The first case of this type was presented by Graham Little at a meeting of the Royal Society of Medicine in 1915. This patient showed numerous areas of atrophy and alopecia of the scalp and an eruption of horny follicular papules on the body. He termed the disease folliculitis decalvans et atrophicans, a bad term and source of subsequent confusion. First of all, the patient had no follicular pustules which are essential for a diagnosis of folliculitis decalvans and second, the picture was merely that of lichen planus with atrophic and follicular lesions both on the body and scalp. In 1924 Little reported another case similar to the one he had reported previously, but in which there were a few definite lesions of lichen planus. He corrected his initial report by suggesting that all of the lesions might be regarded as lichen planus. In 1926 and 1930 he reported several additional cases and connected all of the cases of so called folliculitis decalvans and lichen spinulosus with lichen planus. Feldman also regarded the entire picture as lichen planus, but of a special type which he termed lichen planus

et acuminatus atrophicans. He further linked this disease with lichen sclerosus et atrophicus. His basic findings and conclusions were justifiable except for this observation, inasmuch as lichen sclerosus et atrophicus is a different entity. Montgomery and Hill, in their detailed study of lichen sclerosus et atrophicus, agreed that folliculitis decalvans and lichen spinulosus of Little is probably a follicular form of lichen planus but expressed the opinion that the disease is not lichen sclerosus since histologically it does not show the features of the latter, except for follicular keratotic plugs.



Fig. 252—Follicular plugging and cicatricial alopecia of the scalp. Although such lesions have been diagnosed as *keratosis pilaris* they are usually follicular and atrophic forms of lichen planus. (Courtesy Dr. Marion Sulzberger.)

In 1941 Ellis and Kirby Smith again considered the question of lichen planus et acuminatus atrophicans. Of their three cases, one presented no lesions on the scalp. The second patient presented typical lesions of lichen planus as well as acuminate papules on the wrists and neck and an erythematous atrophic area of alopecia on the scalp 2 cm. in diameter, at the periphery of which were dilated and plugged follicles. No biopsy was performed. The third case presented well defined discrete and confluent bald areas 1 to 2 cm. in diameter, in which the skin was smooth atrophic and slightly depressed. The authors stated that the lesions were typical of pseudopelade. Actually they meant that there were associated patches

of cicatricial alopecia in the scalp. The onset of baldness was accompanied by a generalized eruption and at the time of examination three years later, there were follicular papules on the abdomen and thighs. The histopathologic findings of the lesions on the scalp and body were similar, consisting of well defined bands of lymphocytic infiltration closely packed around the lower ends of the follicles. The histologic changes were identical with those outlined by Feldman as diagnostic of lichen planus et acuminatus. These findings demonstrated that certain (atrophic) follicular forms of lichen planus just as other cicatrizing processes such as lupus erythematosus and favus could simulate the clinical picture of pseudopelade or folliculitis decalvans. The histopathologic findings in this form of lichen planus, however, differ from those of true pseudopelade.



Fig 253—Follicular and atrophic lichen planus of the scalp (Courtesy Dr Marion Sulzberger)

Sachs and De Orco in an extensive study concluded that lichen planopilaris, lichen planus et acuminatus atrophicans (Feldman), lichen spinulosus and folliculitis (Little) were the same disease. According to these investigators most patients give a history of gradual involvement of the scalp and skin over a period of several months (or even one or two years). In some the scalp may be affected first and in others the glabrous skin. Subjective symptoms are usually mild. On the scalp there usually appear two or three or even as many as ten to twelve patches of alopecia varying from 0.5 to 3 cm in diameter. Occasionally these patches coalesce to form a palm sized lesion. The skin of the involved area is thickened, the color may be normal, slightly erythematous or violaceous. The typical flat topped

papules of lichen planus are not usually seen on the scalp. Instead, in the majority of cases, there are acuminate follicular lesions scattered over the involved area but more numerous at the margins of the zone of alopecia. An almost constant observation is the presence of minute keratotic plugs and pin point sized shiny depressions marking the sites of the follicles.

On the glabrous skin Sachs and De Oreo grouped the lesions into two types (1) Typical papules of lichen planus, when present, were found in the usual locations (neck, wrists, and ankles) and involvement of the mucous membrane was noted. (2) All patients showed follicular lesions on the body. When the body hair was involved, the picture was similar to that seen on the scalp. The pin-point-sized shiny depressions were consistently found on the body, though they were more prominent at the sites of adult rather than lanugo hair. The areas of predilection for the second type of lesion were the neck, shoulders, chest, and extensor surfaces of the arms and legs, though any part of the glabrous skin may be so affected.

From an analysis of these observations, it is apparent that there exists a group of cases in which cicatricial alopecia of the scalp with or without marginal follicular plugs is associated with a follicular, papular, lichenoid eruption of the body. These lesions are actually follicular and possibly atrophic forms of lichen planus. The best term, accordingly, is follicular lichen planus. It must be understood that cicatricial alopecia in its final or end stages is often clinically similar whether due to pseudopelade or folliculitis decalvans or to cicatrizing processes following other diseases such as lupus erythematosus, morphea, favus, or atrophic forms of lichen planus. Occasionally, even at this stage, the histopathologic findings may aid in diagnosis.

Pathology

In the common type of lichen spinulosus as seen in children, there is marked keratotic plugging with widening of the orifices of the hair follicles and sweat ducts. There is also some keratotic plugging independent of either. The plug distends the follicle and the upper third may extend above the level of the epidermis. There usually is loss of the keratohyalin layer. There may be a slight increase in the connective tissue cells at the neck of the follicle and about the papillary vessels. The sebaceous glands are atrophied or absent. Gans reported the presence of a mild degree of perifolliculitis.

According to Sachs and De Oreo, the flat and the follicular type of lesion show the same basic picture. An increase in the granular zone, platelike acanthosis, a well defined band of small round cells, and dissolution of the basal margin (observations which make the microscopic picture of lichen planus characteristic) were seen in all of their specimens. Several sections showed Joseph's lacunas.

The interesting feature exhibited by this specific disorder was in connection with the process as it affected the hair follicles. All the essential changes seen in lichen planus were found where the follicles were involved. Hypergranulosis, acanthosis, and a well defined zone of small round cells which invaded the basal margin. In addition there were alterations of the normal structure of the follicle (particularly the papillary portion). The infiltration uniformly pressed against the base of the follicle so that the papilla was obliterated. As the source of blood supply

to the hair bulb comes through the hair papilla it may be that the pressure was sufficient to interfere with the nutrition of the hair bulb and thus produce ischemia and the eventual loss of that structure. This mechanical interference with the blood supply may account for the loss of hair.

These investigators also performed elastic tissue stains of sections from this disease and of sections from ten cases of unquestioned lichen planus, for purpose of control. Their examination showed absence of elastic fibers within the infiltration and some clumping and fragmentation at the periphery and particularly in the neighborhood of the follicles. Such changes in the elastic tissue are not indicative of atrophy and they state that the application of the term atrophy to this dermatosis is based on clinical assumption, not histologic fact.

Etiology

The cause of this disease is unknown. It may possibly be related to avitaminosis A.

Therapy

The treatment is similar to that employed for other forms of lichen planus. In addition, large doses of vitamin A (150,000 units daily) and E (200,500 mg weekly) should supplement such measures. Patients under treatment should be observed for signs of hypervitaminosis A.

Locally, the scalp should be washed twice weekly with a tar shampoo followed by anunction of a keratolytic ointment such as:

Salicylic acid	50
Liquor carbonis detergens	10
Lanolin	750
Petrolatum	q.s. 1000

25 LIPOMILANIC RITICULOSIS

This is the title given to a syndrome consisting of a long standing pruritic dermatosis associated with generalized pigmentation, adenopathy, and eosinophilia. The scalp often shows a diffuse alopecia.

26 PARAPSORIASIS

The various forms of this disease practically never involve the scalp. This fact may be of value from a standpoint of differential diagnosis.

27 PEMPHIGUS

This severe and usually fatal disease not uncommonly affects the scalp. In the vegetating forms the scalp may even be primarily involved, but as in the other types of pemphigus it is usually just another site involved by the typical bullous lesions. In foliaceous pemphigus the scalp is erythematous and scaly, but alopecia is a very late phenomenon since the hairs usually remain tightly attached to the follicle until the patient is practically moribund.



Fig 254—Pemphigus of the scalp. (Courtesy Dr Marion Sulzberger.)



Fig 255—Brazilian pemphigus with involvement of the scalp. (Courtesy Sutton and Sutton Handbook of Diseases of the Skin. Original photograph by Dr O G Costa.)



Fig. 256—*Pemphigus vegetans* of face and scalp (Courtesy of Dr. F. Combes)

28. *PITYRIASIS ROSEA*

This mild disease of unknown origin rarely affects the scalp of the average adult. However, it is an almost invariable location of the disease in children. This fact is not generally recognized and most reports would indicate that scalp involvement occurs in only a minor proportion of the cases of *pityriasis rosea* in children. However, this is far from being the case and in fact, to the contrary, the occurrence of this infection on the scalp must be said to be an almost constant characteristic of the disease. In twelve cases examined by Haxthausen, no less than eleven of these presented a distinct eruption involving the scalp. These twelve patients were all children under the age of 15 years. The reason for the lack of recognition of eruptions thus situated is due to the difficulty encountered in their diagnosis. The usual lesion is not the typical fawn-colored oval patch but is merely a small scaly area scattered irregularly throughout the scalp. As a rule these patches are not hyperemic and correspond roughly in size and shape to lesions on other portions of the skin. Close observation will usually reveal a characteristic circinate collar of scales surrounding the central part of the patches. The scalp lesions are suggestive of *trinea capitis*. In this condition as well as in any other scaly dermatosis involving the scalp of the child, it is imperative that ringworm of the scalp be considered the *working diagnosis until exclusion is specifically performed by adequate laboratory studies*. In such instances the two diseases may be differentiated by the negative

microscopic findings of both the scales from the patches and the involved hairs. In addition, the hairs are not involved in this disease and are entirely normal as differentiated from the brittle and broken stubs and shafts of tinea capitis. Examination under Wood's filter will also bring to light the usual cases of *Microsporum* infection of the hair, although it must always be remembered that not all fungus infections of the scalp impart fluorescence under Wood's filter.

It is interesting to speculate as to the reasons for the localization of pityriasis rosea on the scalp. Its occurrence primarily in children would lead one to suspect that there is some interrelationship between this disease and fungus infections of the scalp inasmuch as the latter too primarily involves the scalp of children before the age of puberty. It is quite possible that the same protective influences (e.g., endocrine, metabolic) are not established prior to that age. The hypothesis which is occasionally advanced linking pityriasis rosea to the various fungus infections is not tenable at the present stage of our knowledge.

Therapy is of little value in this disease. The patches on the scalp as well as those on the body usually disappear within six to eight weeks regardless of the method of treatment employed. In the occasional instance, involution of the lesions may be hastened by the application of exfoliating preparations or by erythema doses of ultraviolet light. In my experience, this may terminate the disease process more rapidly, but will also increase the discomfort of the patient. A soothing lotion or powder will suffice for skin comfort and a bland ointment is adequate for the scalp.

29 PITYRIASIS RUBRA PILARIS

Clinical Features

This lesion often commences as a dry, scaly, seborrheic dermatitis of the scalp. On other occasions it will be ushered in by a fine, scaly desquamation of the face and ears or of the body. This scale is rather characteristic in that it is dry, thin, white, and firmly attached so as to suggest the glazed crackle ware appearance of aged porcelain. Even more characteristic are the small acuminate, firm papules which appear at about the same time. These papules are confined to the mouths of the hair follicles and each is pierced by a hair. At the apex a horny plug or scale dips into the follicle. By coalescence the papules form reddened, scaly psoriasiform areas which may spread and cover the entire body, but at the borders of these areas, or on the dorsa of the fingers, the diagnostic papules with the nutmeg grater feel simplify the diagnosis. Other typical features are the eventual heavy mortartable dirty scaling of the face which gives it a plaster cast appearance and the thick, reddish yellow keratodermic sandals of the soles (and palms as well).

The scalp at first presents the appearance of a mild or dry form of seborrheic dermatitis. In time the scales and crusts accumulate to such an extent as to form a thick, tightly adherent skullcap. Over a period of years the hair diffusely thins out.

Pathology

In this disease the epidermis shows pronounced degree of hyperkeratosis, especially of the follicular orifices. The granular layer is usually hypertrophic but may be thin or absent. Parakeratosis in spots and especially around the hair follicles

is also present : An irregular type of acanthosis is usually encountered, with increased pigmentation of the basal cell layer

In the corium there is mild perivascular and perifollicular infiltration composed of lymphocytes and connective tissue cells with occasional plasma and mast cells : The connective tissue itself shows little if any, change : The sebaceous glands may be atrophied

Etiology

The cause of this disease is unknown

Therapy

In view of the basic follicular hyperkeratosis large doses of vitamin A (150 000 to 300 000 units daily) have been employed with distinct improvement in this disease : In cases associated with hypothyroidism thyroid extract proves a valuable adjuvant to therapy : The local treatment of the scalp is similar to that employed in other locally exfoliative diseases such as psoriasis (page 446)

30 PSORIASIS

Clinical Features

Psoriasis is a strange and capricious cutaneous disorder : It may be described as a chronic occasionally acute inflammatory disease characterized by the appearance of red sharply defined patches on the extensor surfaces of the body particularly the knees and elbows as well as on the scalp the narial regions and the chest : The primary lesion of psoriasis is a small sharply defined slightly elevated papule usually covered with delicate silvery white scales : Upon removal of these scales a bright red surface with minute bleeding points is the pathognomonic characteristic of the disease

The scalp is frequently involved in psoriasis and is sometimes the sole feature of the disease : In the usual instance psoriasis of the scalp is similar in appearance to psoriasis elsewhere on the body : In other words the scalp lesions are flat or only slightly elevated red sharply defined patches of varying size covered with loose silvery scales : These patches may affect any region of the scalp and are often extensions of the eruption from the forehead or postauricular regions : In many instances the diagnosis is enhanced if the area of skin next to the hairline on the forehead is involved thereby resulting in a sharply margined bandlike patch along the anterior hairline : In patients with alopecia due to other causes the eruption shows a predilection for the hairy parts and usually avoids the bald areas : This type is also often associated with erythematous scaling patches about the external auditory meatus

A second and less common form of psoriasis of the scalp is that in which small crusted elevated yellowish papules are scattered throughout the scalp : This type is likely to occur as the scalp component of the sudden showers of lesions observed during acute exacerbations of the disease : Less commonly a third variety of psoriasis of the scalp may be encountered : In this type the entire scalp may be involved with either a diffuse dry scaling erythema or covered by a thick heavy scaly yellow

low crust. The typical silvery scales are much less evident on the scalp in these instances, in fact, the eruption more closely resembles an extensive seborrheic type of "cradle cap."

Although alopecia does not commonly result from psoriasis, these cases may show varying degrees of permanent baldness. Alopecia is even more frequent in psoriatic patients with associated seborrheic dermatitis. It is noteworthy that in psoriasis of the scalp, the hairs, although lusterless and dry, pass through the scales and are not easily removed by traction, as distinguished from fungus infections of



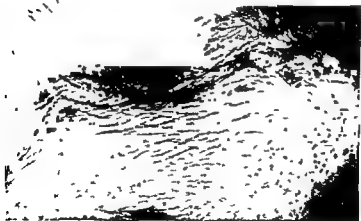
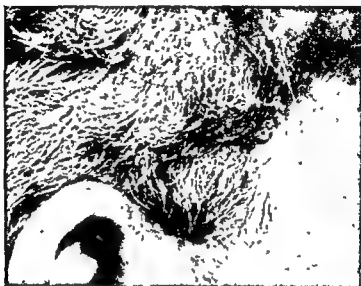
Fig 237—Psoriasis with typical patches on face, scalp, and ears. (Courtesy Dr. F. Combes.)

the scalp and various inflammatory processes. As has been mentioned, there is usually no tendency to hair loss, but this fact has been overemphasized. Long-standing psoriasis, especially if associated with inveterate, thick, heavy scaling patches, may eventually result in alopecia of the affected sites.

Psoriasis of the scalp is not infrequently confused with neurodermatitis and seborrheic dermatitis. This is especially true in those instances where psoriasis may show an associated palm-sized patch on the occiput, which may be thick and prur-

ritic. As a rule, there is little difficulty in distinguishing these diseases from one another. The distribution in psoriasis is on the extensor surfaces, especially the elbows and knees, whereas neurodermatitis affects the flexural aspects of the same locations, and seborrheic dermatitis shows a predilection for the eyebrows, nasolabial folds,

A



B

Fig. 258—A Psoriasis with minimal involvement of scalp and ears. B Psoriasis with minimal involvement of scalp.

postauricular areas, anterior chest and lower back. The scales in psoriasis are silvery white, shiny and dry, whereas those in seborrheic dermatitis are greasy, yellowish, and lusterless. In neurodermatitis the patches are pruritic, thickened, and



Fig 259—Psoriasis with extensive, silvery scaling of the postauricular regions. This is not a common site but may be involved in extensive cases.



Fig 260—Involvement of the scalp in psoriatic erythroderma.



Fig. 61. Psoriasis of the scalp in a full-blooded Negro. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Dr. A. Benson Cannon.)



Fig. 267. Psoriasis with typical sharply defined plaques covered with silvery scales. Despite the frequently repeated statement to the contrary, severe forms of psoriasis of the scalp not uncommonly cause alopecia. (Courtesy McCarthy. Diseases of the Hair.)

lichenified. Removal of the scales from a psoriatic patient results in an oozing of blood from the capillaries. Lichen planus may occasionally show scaling, pruritic and thickened patches in the scalp, but also shows typical violaceous, lichenoid papules over the forearms, wrists, and ankles. In addition, the patches are of a dirty brownish color, the scaling is not at all micaceous, and the margins of the patch often show the typical flat topped papules surmounted by radiating, white striae.

Pathology

Ormsby and Montgomery stated that the histologic diagnosis of psoriasis usually can be made if a well developed papule is selected for biopsy. In the usual instance, psoriasis of the scalp shows a regular acanthosis of the epidermis, a thinning of the suprapapillary plates, and parakeratosis. In the parakeratotic layer, microabscesses are encountered. The corium shows an elongation of the papillary bodies, a dilatation of the capillaries, and a nonspecific, perivascular infiltration of small round cells. Laymon's studies indicated, however, that psoriatic lesions of the scalp do not constantly exhibit the histologic changes regarded as characteristic. He found that hyperkeratosis and parakeratosis were present in every case, although there was extreme variation in intensity. In some cases there was practically no hyperkeratosis, while in others it was extreme. Parakeratosis was present in every case, although in about half it was not uniform but spotty and slight. Almost without exception there was a great diminution in the thickness of the stratum granulosum. In some cases it was uniformly absent, while in others it was present only in small, localized areas. Certainly in psoriasis this layer was more conspicuous by its almost complete absence than in circumscribed neurodermatitis. Generally speaking parakeratosis was present more regularly and extensively in psoriasis than in neurodermatitis, although the difference was not so great as to afford an important differential point. Acanthosis, which ranged from mild to rather intense, was present in most cases and was more regular than in neurodermatitis with the rete pegs more uniformly elongated and clubbed. In Laymon's opinion, the amount of intercellular edema in the epidermis was less than in neurodermatitis, although this point alone was not an important help in differential diagnosis. The number of mitoses in the epidermis and the presence or absence of pigment seemed to be of no value in differentiating psoriasis from neurodermatitis. Microabscesses were not always found in psoriasis but were never observed in Laymon's cases of neurodermatitis.

Just as in neurodermatitis the changes in the cutis were nonspecific and non diagnostic. In some cases no edema was seen while in others it was present to a mild degree. In practically every case the infiltrate was moderate and for the most part arranged about the vessels. There was no special localization about the hair follicles or sebaceous glands. In one case the infiltrate was intense and diffuse. The cells were mostly lymphocytes, although monocytes and plasma and mast cells were occasionally seen. Eosinophils were rarely found.

Laymon agrees with Burks and Montgomery that the histologic differentiation between chronic plaques of psoriasis and plaques of localized neurodermatitis in

the scalp is most difficult. The authors emphasized that both diseases present regular acanthosis and papillomatosis with long and narrow rete ridges and thinning of the suprapapillary plates. In a chronic quiescent psoriatic plaque, microabscesses often are absent or few and far between, and the infiltrate is composed chiefly of mononuclear cells similar to those seen in localized dermatitis. Burks and Montgomery mentioned that dilatation, tortuosity, and apparent rigidity of the walls of the capillaries usually were found in psoriasis and were absent in neurodermatitis. Laymon was unable to note this difference. He believes that the differential diagnosis between the two dermatoses is especially difficult when they occur on the scalp.

Etiology

Although many theories have been advanced the cause of psoriasis is unknown.

Therapy

Psoriasis of the scalp is difficult to treat. In some instances a comparatively simple form of the disease on the scalp may resist all methods of local therapy. On the other hand, long standing and thickened patches of psoriasis on the scalp may respond very well to proper measures.

In acute forms of psoriasis of the scalp associated with similar lesions on the body it is advisable to initiate treatment with bland local applications. This also applies to acutely inflamed patches of chronic psoriasis. The scalp should be shampooed with an ordinary soap shampoo or detergent twice weekly. Daily the scalp should be gently massaged with an ointment such as boric acid ointment, plain vegetable or mineral oil, or 5 per cent liquor aluminum acetate in equal parts of anhydrous lanolin and petrolatum. As the acute phase subsides the therapy employed for chronic cases may be initiated.

Chronic long standing patches of psoriasis on the scalp require constant and skillful therapy. The scalp should be shampooed twice daily with either tincture of green soap or an efficient tar shampoo. Following the shampoo the hair is parted and any one of the following formulas may be employed:

- (1) (Jadassohn)

Liquor carbonis detergens	2 20 0
Ammoniated mercury	5 10 0
Lanolin	50 0
Olive oil	10 0
Water	qs 100 0
- (2) (Sabouraud's favorite prescription for psoriasis of the scalp)

Deodorized oil of cade	10 0
Lanolin	10 0
Petrolatum	10 0
Pyrogallol acid	1 0
Mercuric subsulfate (yellow)	1 0
and	
Neutral washed coal tar	6 0
Lanolin	6 0
Petrolatum	20 0

Ammoniated mercury	10
Resorcin	10
Clear off in morning with	
Alcohol 90%	35 0
Acetone	35 0
Water	qs 100 0
(3)	
Pyrogallol	50
Salicylic acid	30
Pine tar	100
Lanolin	250
Petrolatum	qs 100 0
Pyrogallol discolors light hair	
(4)	
Anthralin	0.1-1.0
Water washable base	qs 100 0
Anthralin discolors skin and light hair	
(5)	
Ammoniated mercury	5 10 0
Acid salicylic	50
Lanolin	300
Petrolatum	qs 100 0
(6)	
Liquor carbonis detergens	5 50 0
Acetone	
Alcohol	equal parts qs 100 0
Paint on patches morning and night	
(7)	
Salicylic acid	100
Pyrogallol	100
Pine tar	150
Lanolin	250
Soft soap	qs 100 0

It is imperative that the scalp be as clean as possible and free from scales before these applications are made. In some instances I have found it extremely helpful to apply the ointment first and massage it in thoroughly and then follow with the shampoo. This is particularly important in women, who object to the matted and tangled appearance of the scalp following the application of an ointment. This problem is circumvented by shampooing out the ointment within an hour following its application and for a time the patient will perform this routine diligently twice daily. Not infrequently the patient cannot perform these instructions carefully and the use of a skilled office nurse or physiotherapist is invaluable. In many instances a routine performed by the patient at home is unsuccessful whereas the same measures performed diligently by a nurse in the physician's office will result in disappearance of the lesions.

The drugs of greatest value in the treatment of psoriasis of the scalp are ammoniated mercury, salicylic acid, pyrogallol, Anthralin and the various tars, espe-

cially liquor carbonis detergens. It is usually advisable to initiate treatment with weak concentrations or combinations of these drugs, and gradually increase the dosage watching the scalp carefully for signs of local irritation. Chrysarobin is not usually employed on the scalp because of its disagreeable staining tendencies on the surrounding skin and the possibility of irritation of the eyes. Although pyrogallol and Anthralin have similar staining properties, they are less obnoxious and less difficult to use if the patient is properly instructed. In order to minimize soiling of sheets and linens the patient should either use old linens or cover the sheets and pillowcases with an old towel or squares of an old sheet. Although patients will often refuse to employ a greasy or malodorous salve on the scalp, instruction in its proper application and the more frequent use of a shampoo may eliminate their objections. In some instances it is advisable to apply an ointment for longer periods of time. In these cases the salve may be thoroughly massaged into the scalp at night and shampooed out in the morning. The patient is then allowed to use a lotion such as the following for daytime application.

Bichloride of mercury	0.1
Resorcin monoacetate	3.0
Silicylic acid	1.0
Spirits lavender	15.0
Castor oil	5.0
Cologne water	30.0
Alcohol (50%)	qs 100.0

Pyrogallol may also be made up in the form of a lotion or varnish and is of considerable value in patients with resistant extremely scaly patches and plaques in the scalp.

Pyrogallol	5.10.0
Ether or acetone	5.0
Alcohol	qs 100.0

It should be reserved for those intractable lesions on the scalp that do not respond to other simpler and less irritating methods of treatment. In this respect it is comparable to chrysarobin in its application in that the latter is also reserved for resistant chronic examples of psoriasis on the body.

As far as the physiotherapeutic measures are concerned the application of the various forms of ultraviolet therapy is of value in psoriasis of the scalp. These measures should be employed following shampooing of the scalp and removal of the scales but prior to the application of an ointment. In the occasional instance a type of modified Goeckerman treatment on the scalp may be quite successful. An ointment containing 2 to 10 per cent crude coal tar in petrolatum or an alcoholic solution of coal tar or liquor carbonis detergens is applied to all the lesions in the scalp at night. In the morning the excessive ointment is shampooed out of the scalp and the lesions exposed to ultraviolet rays. A small cold quartz type of applicator is a convenient method of application but is not as efficacious as the stronger and more penetrating rays of a mercury quartz burner.

Although the judicious application of x rays to limited patches of psoriasis of the scalp will often produce improvement and even disappearance of the signs of

the disease the author does not usually advise the use of this modality. Even small doses of radiotherapy to the scalp may occasionally be followed by alopecia. In addition, despite the fact that psoriasis of the scalp is not usually associated with alopecia, the latter does occur and the use of x-ray therapy is fraught with medico-legal implications. Grenz ray therapy may circumvent this disadvantage. Again a disease as capricious and recurrent as psoriasis of the scalp is best approached from the standpoint of long range therapy with minimal danger.



Fig. 263—Extensive psoriasis with x-ray alopecia of scalp (Courtesy Dr. Frank Coombs)

Internal measures are of as questionable value in the treatment of psoriasis of the scalp as of other portions of the body. I have personally employed low fat diets, autohemotherapy, hypotropic hormone administration, large amounts of all vitamins (A, D, E), estrogens, citrin and undecylenic acid without observing any conclusive benefits. Although cortisone and ACTH have been of value in some instances, their usage is still experimental as far as this disease is concerned. Occasional benefit from these drugs has been reported in extensive cases of arthropathic

psoriasis but it is the arthritis and rarely the psoriasis which improves. Aminopterin has recently been reported of value in the arthropathic forms of the disease with clearance of the psoriasis as well, but further studies are essential for confirmation.



Fig. 264—Severe form of acne vulgaris with pyodermic and cystic lesions in postauricular regions and scalp margins. (Courtesy Clay Adams Co.)

31 SATELLITE OR ABERRANT LESIONS

Satellite lesions on the scalp of a minor degree and similar to those characteristic of the disease elsewhere on the body occasionally appear during the course of the following diseases: acne vulgaris,acrodermatitis continua, dermatitis herpetiformis, dermatitis vegetans, erythema multiforme, exanthems, hydroa aestivale, generalized herpes simplex (Kaposi's varicelliform eruption), pemphigus, Rocky Mountain spotted fever, and urticaria pigmentosa.

32 URTICARIA

The scalp is not uncommonly involved in this disease. The diagnosis is simplified by the presence of typical lesions elsewhere on the body. The primary lesions or wheals are difficult to recognize as such in the scalp and are usually observed merely as small pruritic erythematous macules or plaques, occasionally tense and elevated. The confluence of several lesions may result in a large, tightly stretched and extremely pruritic region of scalp. The treatment is similar to that of urticaria elsewhere on the body, plus the use of various local applications as described under the heading of Pruritus (page 401).



Fig. 265—Urticaria pigmentosa. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Drs. A. P. Biddle and R. Wollenberg.)



Fig. 266—Necroderma pigmentosa with lancelet carcinomas in a child. (Courtesy Sutton and Sutton. Handbook of Diseases of the Skin. Original photograph by Dr. L. Halberstaedter.)

33. URTICARIA PIGMENTOSA

The typical yellowish or reddish brown macule, wheal, or nodule encountered in this disease may also be present on the scalp. In fact, the lesion in the scalp may be so yellowish in color as to be suggestive clinically of a xanthoma and, if the lesions on the body are few in number, is often so misdiagnosed.

34. XLRODERMA PIGMENTOSUM

This rare heredofamilial disease first manifests itself in childhood on the exposed parts of the body (face, upper extremities). It is characterized by brownish, pigmented spots and warty growths. Later, atrophic spots and telangiectases make their appearance, simulating chronic radiodermatitis. The warty lesions usually eventuate in multiple cutaneous epitheliomas with a final, fatal termination. These patients exhibit photosensitivity as a constant symptom and this sensitivity to light may be a causative factor in the progressive development in this disease. The scalp shows a moderate to heavy degree of scaling and may be the site of brownish pigmentation. In addition, the typical atrophic patchy and warty growths may extend into the scalp margins.

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CHAPTER VIII

SCALP INVOLVEMENT DUE TO SYSTEMIC DISEASES

In systemic diseases with skin manifestations, the scalp is merely another portion of the epidermis showing cutaneous involvement. In such instances, the lesions on the scalp resemble those elsewhere on the body surface except for modifications induced by its hairy covering and physiologic variations. Occasionally the presence of such lesions clarifies a diagnosis otherwise obscure, and it is unfortunate that the scalp is so frequently neglected in routine medical examinations. Even jaundice may first disclose its presence by an initial yellowing of the ordinarily white scalp.

Alopecia is also observed in association with various systemic diseases. This may be caused by local involvement of the hair follicle by the disease per se, or by some interference with the blood supply or physiologic balance of the pilosebaceous apparatus by the disease process. High fever (above 103° F) from any cause may lead to transitory or extensive diffuse hair fall within two weeks to two months following the febrile episode. In the presence of local scalp disease, especially seborrheic dermatitis, the hair fall is usually more extensive and slower to return, and occasionally quantitatively less hair is permanently regrown. For this reason, vigorous local measures as outlined under the therapy of seborrheic disease should be conscientiously followed during such periods of illness.

Discoid lupus erythematosus, circumscribed scleroderma and similar cutaneous affections are described in this chapter, although the questions are still unanswered as to their fundamental systemic or local interrelationships.

CALCINOSIS

Calcium deposits are not uncommon in long standing disease processes or new growths involving the scalp.

HAND-SCHÖLLER CHRISTIAN DISEASE

In this syndrome, there occurs a rarefaction of the bones of the skull and the scalp may be the site of a cutaneous xanthoma.

Other features of the disease include diabetes insipidus, exophthalmos and various growth abnormalities.

LIPOID PROTEINOSIS

This rare syndrome is characterized by peculiar yellowish white plaques on the skin and the oral mucous membranes and is associated with hypertrophic warty lesions of the skin. The mucous membranes of the lips, buccal cavity, and pharynx

show nodular infiltration in addition to these plaques, and these nodular changes in the vocal cords and epiglottis produce a characteristic hoarse voice present from infancy. The tongue is smooth but irregular and eventually becomes wooden and bound down to the floor of the mouth. The scalp rarely shows more than a sparse growth of lusterless hair. The edges of the eyelids are covered with beadlike whitish papules and the eyelashes are absent. The disease is probably a manifestation of some constitutional metabolic disturbance, and many of the patients show a diabetic tendency.

LUPUS ERYTHEMATOSUS (DISCOID TYPE)

Clinical Features

The lesions of discoid lupus erythematosus may become evident on any region of the scalp. It has no predilection for specific sites. The disease seldom begins



Fig. 267—Early lesion of lupus erythematosus showing erythema, scaling, follicular plugging and minimal alopecia. (Courtesy Clay Adams Co.)

with scalp involvement and is therefore rarely limited to that location. Usually when there are many involved areas of the skin and mucous membranes, the scalp exhibits comparatively few, but when only a few lesions appear on the body, many

may be found on the scalp. The bridge of the nose, the lobe and tip of the ear, the cheeks ('butterfly pattern'), the lips ('peeling collodion') and the backs of the fingers are familiar sites of concomitant involvement.

The onset is characterized by minute circinate, deep red macules around the hair follicles. These lesions enlarge slowly, gradually coalescing to form one or more slightly elevated pink or dull red plaques, irregular in outline but well defined. These patches may continue as discrete, individual lesions or they may coalesce or merge through peripheral extension until a fairly large, palm sized area becomes affected. The plaques are often covered by small white or gray scales which are usually dry and stubbornly adherent. When these scales are in evidence they tenaciously adhere to the follicles so that their removal often results in minor bleeding.



Fig. 268—Lupus erythematosus showing cicatricial alopecia, telangiectasis and follicular plugging. (Courtesy Clay Adams Co.)

These lesions may remain superficial and erythematous, but in some cases the central zone of the plaque pales in color and becomes glossy and depressed. Hair fall exposes these patches and reveals the usual elements involved in cicatricial



Fig. 269—Old lesions of lupus erythematosus showing scaling, follicular plugging, and cicatricial alopecia. (Courtesy Dr. Marion Sulzberger.)



Fig. 270—Lupus erythematosus showing somewhat unusual type of cicatricial alopecia. The small, superficial scarring and irregular outline of the patches differentiate them from the syphilitic type of alopecia areata. (Courtesy McCarthy, Diseases of the Hair.)

alopecia The dilated follicular orifices are obviously plugged with gray, grayish-white, and yellow keratotic cones which often become painful, are scratched out by the patient, and then bleed slightly from the damaged follicle. Often, the central zone of the affected area remains pale and shiny but its border is an active, bright red. On the scalp, the lesions are usually more infiltrated than on the face but the borders are less erythematous and less elevated. Telangiectatic vessels traverse the patches themselves as well as their erythematous marginal zones. As the atrophic changes expand peripherally, small, white, smooth, slightly depressed, bald areas appear, but "stippling" of the scar is less common than on the facial lesions. The scar is never puckered, radiate, corded, or deeply attached. The longer the course of the disease, the less hair remains. In some cases a few normal hairs do persist in the region of the hyperkeratotic border. Pustulation and ulceration are extremely rare in this disease.

Differential diagnosis is simplified by two characteristics. The active lesions display, first of all, an angry redness usually combined with scaling, and, second, a dilatation and horny plugging of the follicular orifices. Concomitant lesions in areas not located on the scalp are further aids to correct diagnosis. When, however, lupus erythematosus is limited to the scalp and is in its ultimate stage with only atrophic tissue and permanent alopecia remaining without a knowledge of its history it could easily be confused with pseudopelade. During the course of the disease, this confusion could never have arisen since pseudopelade lacks erythema, follicular plugs, adherent scales and telangiectases, never causes pain, and exhibits swollen glassy sheathed roots upon epilation.

Folliculitis decalvans is the source of more serious confusion since it, too, develops an atrophic permanent alopecia with marginal follicular erythematous macules. The diagnosis (at times when there are no simultaneous facial lesions) rests on the fact that the original erythematous macules are minute points of inflammation surrounding the hair shafts instead of having been pierced by them. In addition, dilated, plugged follicular orifices and telangiectases are invariably present in lupus erythematosus. Furthermore, the minute perifollicular pustules of folliculitis decalvans are rarely observed in lupus erythematosus. In healed favus and the end stages of lupus erythematosus, it is sometimes impossible to make a differential diagnosis even with the aid of pathologic investigations.

Psoriasis does not give rise to cicatricial alopecia. In favus, the areas of cicatricial alopecia are white rather than red and there are associated scutula, yellow crusts, and typical microscopic and cultural findings during the active phase of the disease.

Pathology

Early writers differentiated between lupus erythematosus and pseudopelade under the microscope by noting that in the latter the proportional distribution of the infiltrate is denser around the hairs while in the former it is scattered between the hairs. In other words there is a diffuse as well as a perivascular infiltrate of lymphocytes, small connective tissue cells, and an occasional giant cell located about a disintegrating follicle. This heavier and more diffuse infiltrate was limited to the upper part of the edematous cutis.

A

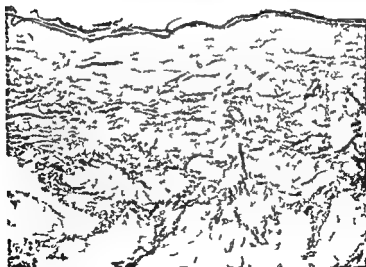


B

Fig 271—Lupus erythematosus of scalp. *A*, Edema and diffuse infiltration of upper cutis. *B*, Same section enlarged to show that infiltrate is diffuse in contrast to pseudopelade. *C*, Same patient. Follicular plugging and dilatation. Diffuse infiltrate of upper cutis. Patchy infiltrate in deep cutis about sweat glands. *D*, Old inactive lupus erythematosus of the scalp. Histologic picture impossible to differentiate from pseudopelade. (Courtesy Dr Carl Laymon and J. Invest. Dermat.)

Histologic study also showed an acute division of elastic tissue in the papillary cutis where fragmentation and splitting existed with not a vestige left visible in the areas of maximum infiltration except for an occasional clump of recoiled elastic

C



D

Fig. 271 C and D (For legend see opposite page)

tissue. The keratotic plugging of both follicles and sweat gland ducts was accompanied by a necrotic plugging of the epidermis with no regard to the follicular orifices and glandular ducts.

In typical lesions of discoid lupus erythematosus, histologic examination may disclose acanthosis, hyperkeratosis, parakeratosis, and atrophy of the epidermis present in the same section. The lower rete is edematous, with areas of basal cell liquefaction. The hair follicles exhibit small horny plugs and these may also be observed between the follicles and independent of them. A slight degree of secondary cyst formation may also be in evidence.

The corium is edematous, especially in its upper region between the areas of infiltration. The infiltrate is present in the neighborhood of the terminal circulation especially around the hair follicles, sebaceous and sweat glands, and the small capillaries. This infiltration is composed primarily of small, round cells with occasional wandering, connective tissue cells. The collagen, especially in the upper corium, often exhibits a basophilic degeneration. Elastorrhexis and clumping of the elastic tissue fibers are also in evidence in the upper corium.

Michelson brought out the fact that the pathologic changes in lupus erythematosus of the scalp are made up of three distinct processes: first, a vascular or perivascular disturbance, second, edema and subsequent damage to connective tissue followed by an infiltration, and third, epidermal and follicular alterations. He emphasized that the changes depended to some degree on the type of lesion under consideration and that a uniform picture for lupus erythematosus does not exist. Laymon observed that the epidermis showed a degree of hyperkeratosis that was mild and insignificant in some, moderate in others, and extreme in still others. Dilatation and plugging of the follicles were present in some cases but were not constantly observed. The granular layer was preserved in almost every instance and in many cases was thickened. Epidermal atrophy was present in almost all instances. In the cutis dilatation of the small blood vessels, particularly in the upper portions, was a prominent feature. Edema was variable. The infiltrate was present in various amounts and in masses of different shapes. Some of the masses were globular, apparently being separated from each other by practically normal connective tissue. In some sections the infiltration was follicular and perivascular, although at many points it was neither. The lymphocyte was the predominant cell, although a few leucocytes were seen. Plasma mast and giant cells were conspicuous by their absence. Deeper in the cutis the infiltrate was rather heavily focused about sweat glands. In most cases of lupus erythematosus of the scalp the sebaceous glands and follicles were destroyed early by the inflammatory process. Staining by the Weigert method showed severe disruption of elastic tissue in the papillary portions of the cutis as manifested by fragmentation and splitting with complete disappearance in areas of maximum infiltration. The infiltrate in lupus erythematosus was distributed entirely differently in active cases than in other types of cicatricial alopecia such as pseudopelade, being much heavier and more diffuse in the upper part of the edematous cutis in lupus erythematosus.

Features of sections from old inactive lesions of lupus erythematosus were entirely nondiagnostic according to Laymon. All that could be said was that the picture was the result of some cicatrizing process. The corium was practically homogenized with absent sebaceous glands, hair follicles, and shrunken sweat glands. The infiltrate, perhaps the chief feature of the active process, had entirely disappeared.

Etiology

Lupus erythematosus of the scalp affects more women than men, and these are usually between the ages of 20 and 45 years. If the disease first manifests itself on the face, only 20 per cent of the men so affected later develop lesions in the scalp but this is true for 50 per cent of the women.

The cause of the disease remains obscure. The French school has persisted in its statement that the disease actually requires a tuberculous soil on which to flourish. Gray's investigation of this pronouncement attempted to prove that lupus erythematosus is actually a clinical entity whose lesions are due to the local activity of a microorganism. Mook and others did not find sufficient evidence that it is the tubercle bacillus which is responsible but did admit that tuberculosis sometimes accompanies the lesions of lupus erythematosus even though it is rarely found in the sanatoria. In England there seems to be a preponderance of patients sensitive to streptococci while on the European continent the tubercle bacillus is more frequently incriminated.

Klemperer, Pollack, and Bachr expressed the opinion that the fundamental damage (*fibrinoid degeneration*) in lupus erythematosus is primarily situated in the collagenous tissue and that apparently the heterogeneous, widespread visceral lesions are merely local manifestations of basic damage to connective tissue. Stokes and his associates refer to a vasculoallergic manifestation and to an infectious allergic mechanism. Montgomery states that lupus erythematosus might be regarded as a disease which affects the reticuloendothelial system, which has a special affinity for the skin, lymph nodes, and various internal organs. He claims that these alterations give rise to toxic disturbances in the body, and decrease the powers of resistance to several types of infection, particularly to infection by the streptococcus and the *Mycobacterium tuberculosis*, and thereby increase the susceptibility of the skin to external and internal influences.

Most authors agree that no matter what the source of infection might prove to be, there are specific predisposing factors such as circulatory disturbances and active trauma. Dissemination frequently follows overenthusiastic removal or treatment of foci of infection and may also occur following exposure to strong sunlight. Fullenbaum concisely stated that lupus erythematosus is a disease provoked by light, attacking a skin sensitized by various bacterial antigens originating far from the actual site of the disorder.

There is still no agreement as to whether discoid lupus erythematosus is an entirely distinct cutaneous entity or is merely one component part of a process ultimately and fundamentally of systemic nature.

Therapy

There are two separate approaches to therapy: the systemic and local, although they may be combined. Careful individual dosage with bismuth or gold salts is the modern method of attack. During this course of treatment, frequent analyses of the blood and urine should be performed.

Gold salts are administered intravenously, usually in the form of gold sodium thiosulfate commencing with an initial dosage of 5 mg weekly and gradually increasing to a total dose of 25 or even 50 mg weekly. Their great disadvantage lies in the possibility of severe toxic reactions to the gold salts.

Bismuth salts are usually introduced via the intramuscular route in doses of 0.2 Gm once or twice weekly. This heavy metal has recently been found equally effective when administered orally in the form of bismuth sodium triglycollamate (Bistriplate). The usual dose is three tablets daily (one tablet equivalent to 75 mg of metallic bismuth).

Good results have been reported following the use of oxophenarsine hydrochloride (Mapharsen) given intravenously in dosage of 0.2 mg twice weekly for a total of sixteen injections. Similar reports following the use of sodium para amino benzoic acid as 0.5 Gm tablets given orally in 2 to 4 Gm dosage every three hours have not been confirmed. There are also favorable reports following the use of mixed tocopherols (vitamin E) in oral doses of 50 to 150 mg daily combined with 400 to 600 mg weekly via the intramuscular route. Savill recommends a combination of quinine and sodium salicylate which she claims acts like magic. In resistant cases the use of Naphuride is sometimes successful. This toxic arsenical is administered in doses of 50 to 100 mg intravenously once weekly, and the patient must be observed very carefully.

The spectacular improvement in acute disseminated forms of lupus erythematosus following cortisone and ACTH therapy have not been duplicated in the chronic discoid forms. The slight improvement exhibited by patients with discoid lupus erythematosus under treatment with these drugs does not justify their use in view of the possibility of severe side effects from cortisone and ACTH.

Sutton believes that one should inaugurate local therapy with mild ointments containing calamine and 0.5 per cent phenol work up through more stimulating applications containing 4 per cent sulfur combined with salicylic acid and finally attempt more irritating remedies containing perhaps 10 per cent ichthammol or pure phenol or a saturated solution of lactic acid. Goldschmid reported enthusiastically on the use of a paste compounded of 10 cc ethyl alcohol added to 1 Gm of potassium permanganate in 10 cc of water to be applied until peeling.

The Hollander treatment is sometimes effective when there are but few lesions on the face and scalp. This consists of the oral ingestion of 0.5 to 1 Gm of quinine sulfate three times daily followed in ten minutes by the local application of tincture of iodine. The treatment is carried out for five to six days then discontinued until the scale comes off and then repeated. I can vouch for its occasional success.

In unusual instances there may be a regrowth of hair on areas of the scalp involved by lupus erythematosus. This is possible of course only if the process is arrested before atrophy has occurred.

In the usual instance of lupus erythematosus of the scalp I prefer to initiate treatment with bismuth salts and vitamin E both orally and parenterally. After a trial period of two to three months unsuccessful cases are then given either gold salts or arsenicals under very careful supervision. Local measures may be of value in early slightly elevated lesions and these are treated with carbon dioxide acetone mixtures or by the Hollander technique. There may be some value in the daily application to scalp lesions of an ointment containing vitamin E (Myopone) with 3 per cent sulfur and salicylic acid. The administration of large doses of vitamin B₁₂ has recently been reported of value in this disease as has the local application of cortisone ointment.

LUPUS ERYTHEMATOSUS DISSEMINATUS

This disease does not usually involve the scalp, although a diffuse alopecia may occur during its course. In very severe episodes associated with extensive cutaneous changes, an occasional hemorrhagic bullous, or crusted lesion may be observed on the scalp. When the process shows concomitant discoid features, the appearance of the lesions on the scalp is similar to that described under the heading of discoid lupus erythematosus. I have also observed an increased hair fall in patients with this disease under treatment with cortisone and ACTH. This scalp hair loss is relatively uncommon whereas an increased facial and body hypertrichosis is seen with comparative frequency in patients treated with these hormones.

PERNICIOUS ANEMIA

The pallor of the skin and scalp in pernicious anemia is usually of a peculiar lemon yellow tint. The hair is fine and often prematurely gray. Vitiligo may be present. A nonspecific eczematous eruption of the neck extending into the scalp margins has been reported. It is probably of a pellagrous type due to an associated avitaminosis.

PORPHYRIA

Congenital porphyria is predominantly a disease of males inherited as a recessive Mendelian trait. The excretion of large amounts of porphyrin ordinarily imparts a dark red color to the urine. Discoloration (usually reddish) of the teeth and photosensitivity of the skin occur. Following exposure to sunlight, the exposed and photosensitized areas of the skin develop bullous lesions. Recurrent episodes of these bullous formations eventuate in scarring and deformity of the affected parts. Hirsutism may be present. Hydroa aestivale is a skin disorder frequently associated with congenital porphyria. It not infrequently produces bullous lesions along the hairy margins.

SCLERODERMA (CIRCUMSCRIBED TYPE)

Clinical Features

Morphea is the name given to cases of circumscribed scleroderma in which the lesions while usually appearing on the trunk are occasionally found on the scalp and its margins.

When the physician is first consulted the early phase of the disease has already passed. A slight itching or burning precedes a mild inflammatory reaction which often continues for weeks or months without alarming the patient. This is followed by the appearance in the scalp of one or several small, erythematous patches which eventually fade out to a dead white color. When the physician finally sees the patient, he is confronted by a glossy hairless slightly depressed plaque of alopecia closely resembling pseudopelade but with two notable exceptions which simplify diagnosis. The white or ivory colored central portion is bordered by a pink or lilac colored areola due to capillary congestion and on palpation one discovers that this central area is as rigid as hardened wax and firmly attached to the apo-

neurosis of the scalp. The thickness of this sclerotic section may either be superficial (measuring about 1.0 mm) or extend more deeply into the subcutaneous tissue (as far as 3.0 mm). Several enlarged blood vessels cross the lesion and similar telangiectases are evident at its margins. At times an exaggerated hypopigmentation appears in the periphery, reminiscent of vitiligo.

The shape of the involved region differs with the individual. There may be one or more discrete patches several centimeters in diameter and either triangular, round, oval, rectangular or linear as when the patch follows the course of a cutaneous nerve such as the supraorbital. In some cases there are clearly defined fingers branching laterally from the original plaque.



Fig. 72. Involvement of the scalp margins in hydroa aestivale, one of the manifestations of porphyria. (Courtesy Dr. Frank Combes.)

The rigidity of the affected area may disappear spontaneously after a considerable time and with its absorption leave behind a localized atrophic patch which becomes the site of a permanent alopecia. The violet border also disappears with the original boardlike consistency making the last stages of morphea difficult to distinguish from the end stages of other cicatricial alopecias unless a history is available of its course from the onset of the initial lesions.

In the scalp the linear or band forms of circumscribed scleroderma show characteristic features as they push their fingerlike projections into its hairy margins. Solitary linear lesions usually extending from the forehead into the scalp resemble the healed wound produced by a deep sharp injury as with a knife or sword and are

appropriately referred to as *en coup de sabre*. The scalp lesion is preceded by a circumscribed depigmentation of the hair, often with alopecia near the median line of the forehead. Following the initial alopecia or depigmentation, the morpheic features appear as a hard marginal elevation with a depressed, sclerotic central trough which gradually enlarges over a period of years. This type of morphea is often associated with facial hemiatrophy as observed by Wartenburg in a comprehensive review. Not only hemiatrophy and scleroderma affect this paramedian area of the scalp, but other congenital malformations of the skin as well. The reason for this fact is that this area corresponds to the vertical line of the body



Fig. 273.—Circumscribed form of scleroderma. (Courtesy Dr. Frank Combes.)

where the bilateral trophic influence of the brain center ceases and the unilateral influence begins. When these centers are disturbed as a result of heredodegenerative processes or inflammatory disease changes of the type described may result. From this discussion it becomes apparent that scleroderma *en coup de sabre* is actually a *forme fruste* of progressive facial hemiatrophy.

In diffuse scleroderma the scalp is usually not involved. In later stages of the disease it may participate in the general hardening of the skin and, as such, is tight and bound down with thin, dry hair.

Pathology

Microscopic investigation shows that the fundamental alterations involved in morphea are those of edema in the early stages with subsequent transition to sclerosis of the connective tissue bundles causing atrophy of the vessels serving the epidermic structures through the application of necrotizing pressure. The adnexa and blood vessels are literally squeezed out. Lymphocytes and large fibroblasts form a damaging perivascular infiltrate. The papillary and dermal vessels become temporarily dilated and ultimately flattened while at the same time the capillaries of the central portion of the plaque exhibit extreme constriction as compared to those of the normal skin. There is less infiltrate to be found in the sclerotic section.



Fig 274 Scleroderma—*en coup de sabre* type (Courtesy Dr Marion Sulzberger)

Concomitant with the constriction of the blood vessels and the condensation of the collagen bundles of the superficial dermis which becomes more homogenous than normal the skin shows a loss of pigmentation and progressive rigidity of the involved plaque. While the connective tissue undergoes sclerosis at an early stage the elastic tissue is retained until the end stages of the process and under the microscope appears elongated and patterned like a coarse fish net.

Etiology

Since the necrotizing process begins with a moderate erythema it would seem that some unknown vascular or neurovascular influence might be responsible for

morphea. Systemic deviations noted during the course of the disease vary considerably and are sufficiently contradictory to confuse rather than clarify the search for a causal factor.

Both high and low blood calcium levels have been reported, and the patients' metabolic rates have ranged from the lowest to the highest. Functional tests have demonstrated vagotonia and sympathicotonia. A few cases have had traces of arsenic in the urine. No endocrine causation has been proved, although adrenal, thyroid, and parathyroid dysfunction have been held responsible by some writers. A consensus may not single out a specific causal factor but rather a group of etiologic components made up of a combination of hypoparathyroid calcinosis, vasomotor disturbance, and endocrine dysfunction.



A



B

Fig 275—Scleroderma—*en coup de sabre* type. A This type may occur in association with hemiatrophy of the face as in this 12 year-old girl who also showed an area of intra cranial calcification. B Same patient showing cicatricial alopecia at the site of the linear sclerodermatous lesion. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photographs by Drs. K. Merritt, H. Faber and H. Bruch.)

Therapy

Many therapeutic approaches have been employed to no avail. All treatment, whether local or systemic tends to be unsatisfactory in the majority of cases. Recommendations include massage, acetylcholine by iontophoresis, niacin, potassium and sodium salicylate, and gold and bismuth given orally and intramuscularly.

Bistrimate has been recently reported helpful in several instances, but this observation has not been confirmed by subsequent studies. Endocrine therapy more or less uselessly attempted includes thyroid extracts, estrogens, posterior pituitary preparations, parathyroid extracts, and even parathyroidectomy. X ray and radium therapy have failed to help.

More recent studies indicate the possible value of various new remedies for both circumscribed scleroderma (morphea) and generalized scleroderma. The neutral sodium or potassium salts of para aminobenzoic acid in daily doses of 1 to 4 Gm. at two hour intervals (total dosage of 5 to 20 Gm.) have been reported helpful in some instances but not in my experience. On the other hand, I have seen several instances in which vitamin E administered orally in doses of 100 to 300 mg. daily and parenterally in doses of 200 to 500 mg. twice weekly, as well as locally to the skin and scalp in ointment form, have shown promise. Cortisone and ACTH have been given with benefit to patients with generalized as well as localized forms of this disease but the improvement requires evaluation as it appears to be of only a temporary nature. Hyperproteinization and androgen therapy have also been reported effective aids in the systemic forms. There have also been encouraging reports following the local use of thorium X in an ointment, varnish, or liquid form. As an ointment the usual dose is 1 000 to 2 000 electrostatic units per gram applied for a 24 hour period and repeated at 4 to 12 week intervals. Such applications would seem to be contraindicated on the scalp.

All these measures may be of no value. My preference at the present time is for the comparatively simple and relatively harmless therapy with vitamin E both locally and internally. In addition massage of the scalp with mild rubefacients (including Mecholyl) is of some benefit.

When associated with generalized scleroderma especially in its early stages a therapeutic trial with the cortical steroid and adrenocorticotrophic hormones and hyperproteinization is warranted.

SICKLE CELL ANEMIA

This disease is a chronic hemolytic anemia, an hereditary constitutional anomaly occurring almost exclusively among Negroes. It is characterized histologically by the crescentic shapes of the red cells which often have elongated as well as pointed ends. At times the cells may be oat shaped or even irregularly stellate. Many reticulocytes and polychromatophilic cells are evident in addition to normoblasts.

The symptoms may include one or more of the following: jaundice, abdominal pain with vomiting, arthralgia with fever, punched out ulcers in the ankle region, heart disease, and neurologic disturbance. Cornbleet and associates reported three cases in which the patients suffered from a concomitant type of alopecia which affected the marginal and lateral regions of the scalp somewhat symmetrical in distribution and extending toward the occiput. The degree of alopecia seemed dependent on the severity of the anemia but once formed it remained without further change. Cornbleet considered the alopecia as an inverse hippocratic type.

Differential diagnosis was not difficult. There was no confusion with alopecia areata because diffuse sparse hairs remained in the affected areas which were

neither ovoid nor circular in pattern. The denuded regions did not alter their shapes nor shift to other localities. Microscopic examination revealed that the remaining hairs were entirely without evidence of nodules, twisting, fraying or exclamation points. Neither direct examination nor culture disclosed a fungus infection. The only difference noted was that the sparse remaining hairs were duller than those from the normal scalp.



Fig. 276—Sickle cell anemia with ophiasis type of alopecia. (Courtesy Drs. Theodore Cornbleet, H. Schorr, and S. Barsky and *Arch. Dermat. & Syph.*)

SJÖGREN'S SYNDROME

This disease is characterized by extreme dryness of the skin and mucous membranes (keratoconjunctivitis sicca, xerostomia). It is presumed to be caused by a combination of vitamin deficiency and endocrine dysfunction. The scalp may be involved, as evidenced by erythema and scaling.

VOGT-KOYANAGI SYNDROME

This rare syndrome is characterized by uveitis, graying of the hair, vitiligo, dysacusis, and alopecia of the type observed in alopecia areata. The entire syndrome may be due to a virus infection, and in severe cases involvement of the eye may lead to blindness. I observed one instance of this disease in which an almost total alopecia was present. Recovery was followed by return of the hair, but the initial black color was replaced by a permanent gray shade.

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CHAPTER IX

NEW GROWTHS

INTRODUCTION

The scalp may be the site of almost any type of neoplasm. Although it is not a common site for a single or primary tumor either benign or malignant, almost any type of neoplasm may be encountered on its surface. When multiple lesions are present on the body, the scalp is merely one part of the body surface which may be involved and show one or several tumors. In their early stages the majority of these growths are small and inconspicuous, often remaining hidden by the hairy covering. Subsequently as a result of increasing size and irritation from combing, scratching, and brushing they are often detected for the first time although they may have been present for many years. Their true nature is often obscured at the time of the initial examination in view of the fact that this preceding trauma results in crust formation thereby masking the underlying lesion. A period of observation and the employment of bland local measures suffice to disclose their identity. Changes in the hair itself are uncommon although alopecia may result because of atrophy from pressure of the tumor mass or as a result of actual invasion and destruction of the pilosebaceous apparatus. Although malignant tumors are not common on the scalp yet they do occur and must be recognized. Emphasis must be placed on the fact that proper diagnosis and treatment administered to the small innocent appearing lesion will often prevent the development of frank malignancy and subsequent metastases. In general it is not necessary to be concerned with the overly careful cosmetic removal of scalp lesions inasmuch as the hairy covering will frequently act as a disguise for subsequent small areas of cicatricial alopecia. Too often the oncologist observes small neoplasms of the scalp which have been treated over a period of years with modalities such as salves, caustics, electrosurgical procedures, and inadequate radiotherapy until a large and extremely serious lesion becomes manifest and requires drastic measures. For accurate diagnosis and proper treatment it is advisable that an adequate specimen of tissue be removed and submitted for histologic examination. If possible this procedure should be an integral part of the first treatment or series of treatments designed to eradicate the growth completely.

GENERAL SURGICAL CONSIDERATIONS

The skin of the scalp is bound by fibrous septa to the galea aponeurotica (the epicranial aponeurosis) which is only loosely attached by areolar tissue to the

underlying skull covering (*pericranium*) The cutaneous, subcutaneous, and galeal layers, therefore, are virtually one and act like a single layer Certain surgical considerations therefore, follow from this anatomic characteristic

1 Infections

Infections beneath the galea aponeurotica can spread from one end of the skull to the other in the loose areolar layer beneath the galea stopping at its attachments—to the superciliary ridges in front and the occipital protuberance behind

Carbuncles of the subcutaneous layer often are difficult to drain surgically by crossed incisions because undercutting and separating the superficial layers from the attached galea are not satisfactory Excision of the carbuncle if small or deep incisions through the galea may be necessary Fortunately antibiotic therapy is usually sufficient to control even the most extensive carbuncular scalp infections without operative intervention

2 Lacerations and Incisions

Lacerations or incisions which do not sever the aponeurotic layer usually do not gape and are easily approximated but the blood vessels running in the thick fibrous septa of the subcutaneous tissue may bleed profusely and fail to contract They are difficult to grasp with an artery clamp and are often best controlled by simple pressure on either side of the wound and by approximation of the wound edges

Lacerations and incisions through the galea aponeurotica may result in wide separation exposing the pericranium and requiring approximation of the aponeurotic layer as well as the skin However sutures including all layers (skin subcutaneous tissue and aponeurosis) are easily passed since the three layers are bound together These deep sutures usually provide hemostasis but sometimes separate interrupted sutures through the aponeurotic layer are a further aid in maintaining hemostasis For the immediate control of bleeding in an incised wound it is necessary to group the galea together with the bleeding point superficial to it for the vessel itself cannot usually be held separately

Blows on the scalp by blunt objects may cause a linear laceration because the inelastic scalp may split

Avulsions are through the subaponeurotic layer leaving the underlying pericranium bare This thin covering of the skull may become necrotic permitting the exposed bone itself to undergo necrosis Replacement of partially avulsed scalp even though attached by only a narrow pedicle may often be successful because of the rich blood supply in the subcutaneous layer

3 Removal of Tumors and Cysts

Sebaceous cysts which do not cause much elevation of the overlying skin may be removed by incision directly over the mass down to the cyst capsule The proper plane may then be entered by sharp dissection with the knife blade held parallel to the skin surface The cyst can usually then be shelled out bluntly If the cyst is entered accidentally it can still be removed easily in the same manner

However, cysts which cause elevation of the skin, producing a 'bump,' are simply removed by making an elliptical incision over the summit. This procedure has several advantages. The proper plane is more easily entered for removal by blunt dissection; a handle is provided for grasping the mass and obtaining traction; the subsequent skin closure is simplified. When a linear incision is employed, excess skin and underlying dead space may be left, allowing for the accumulation of serum and blood.

Lipomas of the scalp are often subaponeurotic in location and require incision through the galea aponeurotica. Bleeding which may be profuse in this process, is often controlled easily by a row of clamps on either side of the incision, grasping the galea and bleeding points together.

4 Local Anesthesia

Infiltration into the skin or subcutaneous tissue is often difficult because of the resistance offered by the inelastic fibrous septa, but the subaponeurotic layer is easily entered and permits deposition of a local anesthetic into its loose areolar tissue. Since the sensitivity of the scalp is not remarkably acute, removal of superficial tumors is often possible with only scant anesthetic infiltration into the dense subcutaneous tissue, but if pain is not controlled by this means, the deeper areolar layer should be entered with the needle.

5 Dressings

Pressure dressings in one area are usually not possible except by including the entire scalp in the bandage head roll, etc. For most superficial excisions, however, a small cloth patch held by liquid adhesive is often sufficient. A very efficient dressing is a collodion dipped gauze patch with a separate dry gauze layer interposed between the wound and the collodion gauze, so that later removal of the dressing will not pull upon the suture.

In view of the confusion and many difficulties encountered in the attempts to classify these growths adequately, the tumors will be presented and discussed in alphabetical sequence.

I BENIGN EPITHELIAL NEW GROWTHS

1 CORNU CUTANEUM (CUTANEOUS HORN)

These lesions are benign horny overgrowths of the epithelium, usually found in older patients. As the name signifies, the lesions appear as straight or twisted horny projections varying from $\frac{1}{4}$ inch to 3 inches. They are often minute replicas of an animal horn. These lesions grow from long standing neglected warts, sebaceous cysts and papillomas. More than 50 per cent of these lesions develop on the sites of a preexistent senile keratosis and not uncommonly undergo malignant change.

The histologic changes depend on the origin of the horn. Inasmuch as this source is usually a senile keratosis, the microscopic findings are those of marked

hyperkeratosis of tightly packed horny lamellae, irregular acanthosis and a moderate, small round cell and plasma cell infiltration in the upper corium. The horn itself is composed of layer upon layer of hyperkeratotic stratum corneum. When malignancy ensues it is most often of the prickly cell variety and may be preceded by Bowenoid changes.

The treatment consists of either simple excision or electrosurgical removal. If histologic examination shows the presence of malignant change, incompletely removed lesions should be followed by an adequate course of radiotherapy.



Fig 277—Cutaneous horn which was 20 cm long and 8 cm in circumference (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Dr H Charache.)

2 EPITHELIAL CYSTS

a Dermoid Cysts (Epidermoids)

These lesions are comparatively rare tumors of the skin related to epithelial and sebaceous cysts. They usually arise along the lines of closure of various embryonic clefts and may be considered as developmental anomalies. The cysts are often present at birth and increase in size during childhood. Their usual location on the scalp is behind or just below the ears although they may occur on the scalp itself. Unlike sebaceous cysts they are not attached to the skin and are freely movable under it and over the underlying fascia. They are also of a firmer consistency than sebaceous cysts. These cysts contain sebaceous material and keratin and often have rudimentary sebaceous and sweat glands as well as hair follicles and mature hairs in their walls. Older cysts occasionally show calcification, bone formation, and even malignant degeneration.

The treatment consists of simple but complete surgical excision. It is important not to leave any portion of the cyst wall in the scalp as subsequent recurrences and malignant carcinomatous change may supervene.

b Keratoma

These lesions are usually found on the scalp as single and occasionally multiple firm nodules varying in size from $\frac{1}{4}$ to 1 inch. They give rise to no symptoms



Fig 278—Sebaceous cysts, well exposed by coincidental alopecia areata. The hair is beginning to return in these patches, and is white as usual. (Courtesy Sutton and Sutton Diseases of the Skin. Original photograph by Dr J P Guéguière.)



Fig 279—Sebaceous cysts of many years' duration. (Courtesy Dr Frank Combes.)

They are frequently confused with sebaceous cysts but can be clinically differentiated by the following facts

- 1 They do not possess a central opening from which cheesy, odoriferous material may be extracted
- 2 They are freely movable in the skin [Sebaceous cysts are usually attached to the skin]
- 3 The lesions are usually smaller in size than sebaceous cysts
- 4 The epithelial cyst is harder and firmer
- 5 " " "

and they do not regard these lesions as precancerous

The lesions may be removed by simple excision

c Sebaceous Cysts

These cysts are commonly encountered on the scalp and are often multiple. They vary in size from that of a pea to that of a small orange. These cysts are attached to the skin and usually communicate with it through a small duct leading to the surface. This central duct is not infrequently blocked by a thick black plug. They are usually attached to the skin but are freely movable over the underlying connective tissue. In time, the enlarging cysts cause pressure atrophy of the overlying skin and destruction of the hair follicles, giving rise to the term 'bald headed cysts'. The skin overlying these cysts is often of a dead white hue due to this pressure. However, in elderly and obese individuals, it may be extremely red and greasy.

Histologically, the wall of the cyst is made up of thick fibrous connective tissue with a lining of flattened epithelium. Sebaceous cysts may be present in the lining of the wall. Unlike the walls of the epithelial cysts, the epithelium of sebaceous cysts does not go on to complete keratinization. The contents of the cyst are composed of cast off epithelium, fat, and cholesterol crystals forming a cheesy and odoriferous material. Calcification sometimes occurs in these cysts. During a routine study of all sebaceous cysts removed at Barnes Hospital, Peden found carcinoma in 14 out of 832 cases. This incidence of 1.7 per cent does not justify considering sebaceous cysts as potential or precancerous lesions.

The best method of treatment consists of simple surgical excision. This is usually done by means of an elliptical excision including the area of attachment of the cyst to the skin as evidenced by the enlarged follicular opening. When these lesions are removed from a hairy portion of the scalp it is difficult to retain a dressing even though the surrounding skin may have been shaved. In these instances it may be kept on by the means of utilizing long strands of hair tied in several directions across the scalp dressings. Small cysts may be removed by puncturing the surface with an electrodesiccating or electrocoagulating current permitting the current to flow until the surrounding tissue shows slight blanching. I have not

been impressed by my results with this technique. These lesions have also been successfully eradicated by careful repetitive injections of small amounts of a sclerosing solution directly into the cyst, and also by the injections of 70 per cent alcohol, aqueous penicillin, bacitracin, and similar antiseptic and slightly irritating solutions.

3 EPITHELIAL NEVI

Benign moles, as these lesions are sometimes referred to, are difficult to classify. According to Montgomery, the term 'epithelial nevi' should be limited to any nevus disturbance, either present at birth or developing later in life, in which hyperplasia of epithelial cells occurs without the presence of nevus cells. When nevus cells are present, he specifically classifies the lesion as a nevus pigmentosus. Although he restricts the term epithelial nevi to linear nevi and hard nevi (Unna), they may all be included under the term of benign moles or epithelial nevi. These lesions are found rather commonly on the scalp as well as on the skin. Although variations may occur ranging from simple areas of increased local pigmentation to verrucous papillomatous and plaque-like elevations, the two most common varieties are the flat type, which may contain hairs, and the smooth, soft, slightly raised, hairy brown nevus. The flat type is often, but not always, a junction nevus, and vice versa. The degree of pigmentation may vary from light brown to blue or black. Approximately 40 per cent of these benign nevi occur on the scalp, face, and neck.

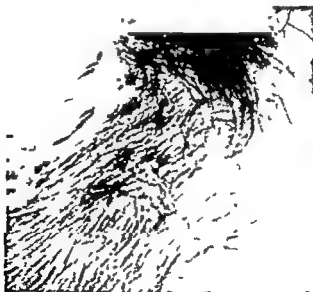


FIG 280—Intraepidermal pigmented nevus

The statement is frequently made that lesions containing coarse hair are either less likely or unlikely to become malignant. This cannot be accepted as a clinical guide inasmuch as the presence or absence of hair is not an indication of the absence or existence of malignancy. It must also be recognized that these cellular nevi may be associated, in the same lesion, with other types of nevi (vascular, glandular, etc.)

Histologically these lesions may be classified under one of three headings

- a **Intraepidermal Nevus**—In this type the changes are limited solely to the epidermis. The epidermis appears as though it had been twisted out of shape and reshuffled into a disorderly pattern. The surface may be irregular and verrucous. It may or may not be covered by the stratum corneum. Melanin may be present or absent. They may or may not contain hair. These lesions are invariably benign.
- b **Junction or Marginal Type Nevus (Melanoma)**—This is the lesion usually referred to as the true pigmented nevus. The nevus cells extend from the undersurface of the epidermis, down to the corium in great masses. Many *cellules claires* (Masson) are present in these masses and are assumed by some to be the origin of the nevus cell. These cellular masses invade the corium in an inverted V shape and for that reason a junction nevus should be removed widely. The lesion may be associated with hair or pigment.
- c **Cutis Type Nevus (Intracutaneous Nevus)**—In this group, microscopic

may or may not contain hair. Malignancy rarely occurs.

Therapy depends on whether or not malignancy may occur. On the scalp the small, flat, unchanging lesions do not usually require any treatment. If, however, the lesion is the site of repeated trauma from combing, scratching or brushing and shows signs of cellular activity as evidenced by subjective symptoms (itching or burning), increase in size or pigmentation, frequent bleeding and crust formation, there is no question as to the importance of a speedy removal. According to Pack, congenital moles have their greatest rate of growth at the age of puberty and may then become malignant without trauma, and he unequivocally states that all moles on the scalp should be removed because irritation is unavoidable. There is little doubt that elevated large and dark moles should be removed before puberty.

It is important for the physician to realize that in children lesions which are histologically malignant may have a relatively benign character. A great deal of discussion has centered about the methods employed for removal. The dermatologist has been criticized because of his preference for the simpler, electrosurgical techniques. The statement has been made, usually by the surgeon, that metastases are more frequent and recurrence is more often found following electrosurgical procedures. This is not true! Recurrences are just as likely to occur following scalpel surgery, and metastases may occur prior to as well as following such procedures. The only importance attached to any procedure is the thoroughness of the local removal. A complete and thorough removal at the time of the initial procedure will insure the best result.

4 KERATOSES

a Arsenical Keratosis

These lesions follow the ingestion of inorganic and pentavalent forms of arsenic and rarely affect the scalp. They resemble senile keratoses both clinically and histo-

logically but are practically always limited to the palms, soles, and fingers. In the very exceptional case, lesions on the face and scalp margins may be present, but the patient presents typical arsenical keratoses of the palms and soles as well. These lesions often progress to squamous cell epithelioma formation.

The preferred method of treatment is surgical excision.

b. Seborrheic Keratosis (Verruca Senilis)

These lesions appear later in life and are actually delayed epithelial nevi. They are usually of a light yellow to deep brown color and slightly elevated, with a smooth, velvety surface. On the scalp these lesions are often encountered just inside the hairy margins of the frontal and parietal regions. The growths are usually multiple and are more frequently noted in individuals with greasy, seborrheic skin.



Fig 261.—Seborrheic keratoses. (Courtesy Dr Richard Sutton, Jr.)

Histologically the seborrheic keratosis shows an acanthosis of a mild to moderate degree. The rete pegs have a tendency to anastomose, producing a lattice-work appearance. Occasionally, horn cysts are observed. The basal cells and their margins show an increased degree of pigmentation. In the corium, there is a very mild, nonspecific cellular reaction. Some basophilic degeneration may be present but is not a constant feature.

Therapy is usually desired because of irritation from combs and brushes. These lesions are easily removed by means of superficial electrodesiccation. According to McCarthy, a good cosmetic result may be secured by means of radiotherapy, but

this is not in agreement with my experience. The lesions are actually radioresistant. This lack of radiotherapeutic response is probably due to a marked degree of differentiation of the cells.

c Senile Keratoses

These lesions are frequently found on the scalp margins, face, neck, and hands of people who live and work out of doors. In many of these individuals the skin shows the extremely dry and wrinkled aging associated with other signs of senile



Fig. 282.—Seborrheic keratosis, clinically suggestive of a basal cell epithelioma.

change. This type of skin is often referred to as farmer's or sailor's skin, inasmuch as frequent exposure to sun and wind often accelerate its development in blue-eyed, light-skinned individuals. The lesions appear on the temples and scalp margins as circumscribed patches composed of brownish, slightly elevated, firmly adherent scales and crusts. Removal of the crusts is frequently followed by bleeding and superficial ulceration. Approximately 15 to 25 per cent of these lesions eventually

develop into squamous cell epitheliomas. When malignancy supervenes, the kerototic lesion appears inflamed around the edges and the base becomes thickened or indurated.

The histologic features consist of marked hyperkeratosis of the epidermis with closely packed horny lamellae. An irregular acanthosis of a moderate to extreme degree may be observed. There are spotty areas of cell disturbance with a separation of the basal cells. Bowenoid changes may also be present in the rete. The corium shows an infiltrate in its upper layers composed of small round cells, plasma cells and connective tissue cells. There is also a basophilic degeneration of the connective tissue. At the dermoepidermal junction a basal cell pigmentation may be observed.

Therapy depends upon the results of histologic examination. At the time of removal of the biopsy specimen the lesion itself may also be removed by means of superficial electrodesiccation. If this procedure has been thorough though superficial a good cosmetic result may be obtained and no further therapy is indicated provided microscopic examination shows no sign of malignancy. If the latter has occurred it may be advisable to re-treat the lesion by means of a more extensive electrosurgical technique. If the area is not too extensive, surgical excision is also satisfactory. Irradiation therapy may be given by roentgen or radium rays. The author has employed the following technique approximately ten days following superficial desiccation providing the lesion is less than 2.5 cm in diameter. Roentgen therapy is administered in doses of 600 r given every other day for a total dose of 3 600 r (120 kv with filtration of 0.25 Cu and 1 mm Al and a half value layer of 7.8 mm Al).

Radiotherapy should be used with caution because of the similarity of the histologic changes of senile keratosis to those of roentgen keratosis. In fact some oncologists do not advise its use in the treatment of this growth. It is essential that these patients be advised to avoid exposure to strong sunlight and local irritative processes of any kind in order to minimize recurrence and the development of new lesions. Protective creams and applications may be necessary if exposure to sunlight is unavoidable. Fifteen per cent para aminobenzoic acid in hydrophilic ointment L.S.P. is a very effective sun screen.

5 MOLLUSCUM CONTAGIOSUM

Although the lesions of this disease are usually encountered on the body they may occur singly or in great numbers on the scalp. They may appear only on the scalp and this occurrence may be more frequent than was formerly suspected and merits consideration in the differential diagnosis of tumors of the scalp. The lesions are small waxy centrally umbilicated firm papules. Because of this waxy appearance with a central dark colored opening they may resemble small pearl buttons. They vary in size from 1 mm to several centimeters. Cheesy material consisting of degenerated epithelial cells (molluscum bodies) may be expressed through the central openings. Inclusion bodies characteristic of virus disease may be demonstrated in some of these molluscum bodies. The lesions rarely grow larger than 1 cm in size on the scalp. Occasionally these small growths become

secondarily infected and may heal without further treatment. Although the scalp is the uncommon site of a single lesion, they have occurred as in the case reported by Hill and Messina. In this instance they reported a pea sized, round, waxy nodule with a rolled border and central hemorrhagic crust over the left parietal region. The diagnosis of basal cell epithelioma of the scalp was made and the lesion excised. Subsequent histologic study revealed the lesion to have been a molluscum contagiosum. Similar cases have been reported by others. Young reported the occurrence of such an eruption in a 2 month old girl, which started on the forehead and extended to the scalp. Kiefer observed two cases in which the lesions were confined to the ocular regions: in one the lesions were confined to the lower lids, and in the other to the conjunctiva. White observed a case in which there was only one lesion, situated on the left side of the neck and another in which a single lesion was present on the scalp.



Fig. 283—Molluscum contagiosum of the scalp (Courtesy Dr. Samuel Peck)

Histologically, the molluscum tumors first appear as small, benign proliferations of the prickle cells which gradually extend into the corium as large pear shaped nodules which flatten the papillae into interlobular septa. The growths subsequently form a cavity near the center and the cavity extends to each of the nodules composing the lesion. In the prickle cells themselves, the nucleus shrinks and is pushed to one side. The cytoplasm of the cells swells and takes a more intense stain. The nucleus eventually becomes completely obliterated. The molluscum cells resemble an eye in that the nuclei of the cells are pushed to one side and the cytoplasm stains very poorly. The molluscum body has no nucleus at all and contains one or many elementary bodies. Strictly speaking the term molluscum body should refer to the eosinophilic inclusion bodies and probably represents the causa

tive virus which may occupy the entire cell : The process does not affect all of the cells, and the involved ones are surrounded by partially and fully keratinized, normal prickle cells

The treatment of this disease can be successfully accomplished in any one of many ways . Perhaps the simplest method is the curettement of the entire lesion followed by the painting of the base with tincture of iodine or 10 per cent silver nitrate solution . Local anesthesia is not usually required since the lesions come away readily with little pain . A local anesthetic is necessary on occasion and preliminary freezing by means of an ethyl chloride spray may be adequate . The lesions also respond to the application of trichloroacetic acid and to insertions of a pointed wooden applicator, the tip of which was previously dipped in phenol . In similar fashion the electrodesiccating needle may be inserted into the lesion and the current permitted to flow for a few seconds . In some instances one or several of the lesions are infected and require preliminary treatment by means of wet dressings . Several recent reports have mentioned cures in extensive cases following the internal ingestion and external application of antibiotics such as penicillin and aureomycin . In my experience these drugs are only occasionally of merit .

New lesions may continue to appear in view of the six week incubation period of the molluscum tumor and the patient should therefore be required to return for observation . Failure to observe this rule is responsible for the occasional recurrence of the lesions . It is also advisable to inform the patient regarding the contagious nature of the disease and measures to avoid autoinoculation and transmission of the disease to others . Towels and linens should be kept apart from those of other members of the household .

6 MULTIPLE BENIGN CYSTIC EPITHELIOMA

(Trichoepithelioma, Epithelioma Adenoides Cysticum)

The distinctive features of this disease are the occurrence of pinhead to pea sized pearly flesh colored papules on the face . When many lesions are present it is not uncommon to find one or several along the margins of the scalp . This disease occurs more often about the time of puberty and is frequently hereditary . These lesions have sometimes been confused with milia or syringoma and may even suggest vesicles . The diagnosis may be clarified by their firm solid nature and the lack of extrusion of any material following the insertion of a needle into the growth . Malignant change rarely eventuates unless the lesions have been constantly irritated . In some instances these lesions resemble syringoma but the latter are found in much greater profusion on the scalp and are considerably larger in size .

Histologically these lesions are characterized by the presence of ropelike epithelial tracts or fingerlike projections in the corium . These masses are composed of basal cells which have arisen from the hair follicle or the basal margins of the epidermis . Oval or round cysts are formed and these are surrounded by dense fibrous connective tissue with practically no inflammatory reaction . In some cases it is not possible to differentiate the features of multiple benign cystic epitheliomas from syringomas adenoma sebaceum and cylindromas . According to some pathologists these lesions are merely variants of the same process .

These growths are difficult to eradicate . When many lesions are present on the scalp and face the best cosmetic results may be secured by means of superficial

electrodesiccation followed by curettement. This procedure may necessitate repetition on several occasions in order to minimize scarring from a single, overenthusiastic technique. It is preferable to perform superficial removals on subsequent visits. Where only a few lesions are present, a plastic excision or superficial electrosurgery will each yield satisfactory results.

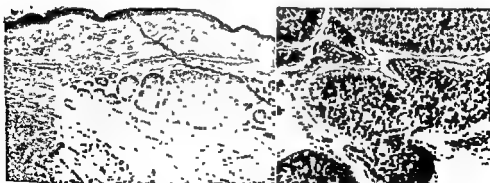
7. NEVUS EPITHELIOMATOCYLINDROMATOSUS

(Turban Tumor)

This group of tumors occurs on the head and neck but more frequently involves the scalp. The lesions usually appear as multiple, lobulated or mushroomlike



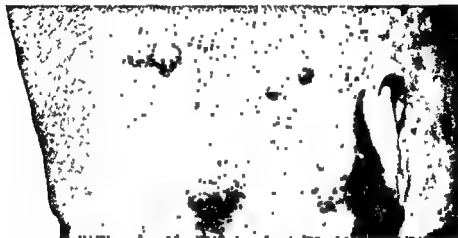
A



B

Fig 284—Nevus epitheliomatocylindromatosus. A, Typical lesions on forehead and scalp. B, Histologic features of multiple benign cystic epithelioma. These lesions may show the microscopic features of syringoma, nevus epitheliomatocylindromatosus, and multiple benign cystic epithelioma with transitions from one to the other. Malignant change is uncommon. (Courtesy Sutton and Sutton. *Diseases of the Skin*. Original photographs by Dr F Ronchese.)

tumors, varying in diameter from 1 to many centimeters. Their color varies from that of normal skin to shades of red or brown. They may either be pedunculated or have a broad base. Alopecia is present over the lesions as a result of pressure atrophy. The tumors slowly increase in number and enlarge over a period of years, often grouping together to resemble a bunch of grapes or tomatoes. In some instances they are so numerous that they cover the entire scalp, giving the appearance of the surface of an Easter basket or turban composed of small eggs. Some authors



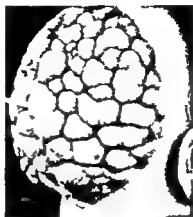
A



B

Fig. 285 — *Nevus epitheliomatocylindromatosus*. A, Single, firm tumors over the posterior scalp. B, Histologic features of masses of basal cells. (Courtesy Sutton and Sutton, *Diseases of the Skin*. Original photographs by Dr. H. C. Varney.)

are of the opinion that there is an hereditary factor and cases have been reported as occurring in several members of the same family and through several generations. It is generally accepted that these tumors are epithelial in origin and begin in the matrices of the hair follicles.



A



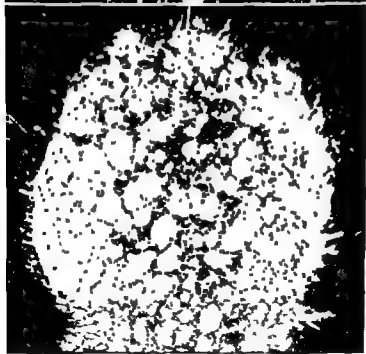
B

Fig. 286. Nevus epithelionatocylindromatosus (turban tumor).

A. Typical appearance of variable sized multiple tumors. (Courtesy Sutton and Sutton. Diseases of the Skin. Original photograph by Drs. H. Hammond and H. Ransom.)

B. Similar lesion with both grouped and single lesions. (Courtesy McCarthy. Diseases of the Hair. Original photograph by Dr. Stillans.)

A



B

Fig 287—A, Cerebriform nevus of scalp (a variant of nevus epitheliomatocylindromatosus) (Courtesy Dr Marion Sulzberger)

B, Close up of a clinically similar lesion (Courtesy Clay Adams Co)

Histologic studies disclose a thinned out epidermis. In the corium there are masses or nests of epithelial cells with deeply staining large, round or oval nuclei and a sparse cytoplasm. The outer row of these cells resemble the basal cells of the epidermis and usually presents itself in a palisade like arrangement. The entire mass is separated from the epidermis by a band of connective tissue and the individual masses or nests are separated by connective tissue septa. Within and surrounding these masses, are hyaline deposits which are actually rings of hyaline degeneration. Some pathologists have stated that these tumors arise from mature sweat glands, but detailed studies have failed to trace any connection between these tumors and either the sweat ducts or the sweat glands, although their apparent origin from the basal cells of the epidermis and from the basal cells of the outer surface of the walls of the hair follicles has been repeatedly demonstrated. In some instances the histologic picture is confusing in that it may show features of multiple, benign, cystic epithelioma and syringoma as well as the typical features of this nevus.



Fig. 288—Nevus sebaceous. The location of this linear growth involving the side of the face and the scalp above the ear, is typical. Note isolated umbilicated yellowish elements at the outer corner of the eye. (Courtesy Sutton and Sutton. *Diseases of the Skin*.)

Therapy is often rendered extremely difficult in view of the multiplicity of these lesions. Inasmuch as malignant change is extremely uncommon, these extreme and advanced cases of turban tumors are probably best left alone unless they show signs of ulceration or degeneration. In such instances, surgical excision is the best method of treatment. Where single lesions are present, recurrences will also probably be minimized by adequate surgical removal. Where removal is essential even though the lesions are many in number and affect large areas of tissue, surgical excision may be employed, followed by plastic repair. These lesions do not respond to radiotherapy.

8 NEVUS EPITHELIOMATOSUS SEBACEOUS CAPITIS

According to McCarthy these lesions occur as unevenly elevated brownish yellow hairless plaques on the scalp. Histologically the tumor tissue is rich in sebaceous glands and shows a proliferation of the connective tissue as well as the production of abortive hairs. These growths are easily destroyed by electrodesiccation.



Fig. 289. Nevus epitheliomatosus sebaceus capitis. (The tumor tissue is rich in sebaceous glands and there is a proliferation of the connective tissue as well as the production of abortive hairs. (Courtesy McCarthy. *Histopathology of Skin Diseases*.)

9 NEVUS SYRINGADENOMATOSUS PAPILLIFERUS

This rare disease occurs on the scalp, shoulders, axillae, and genital regions as minute, firm, red papules often surmounted by pinhead-sized clear vesicles. An occasional papule may be umbilicated. The lesions resemble those of syringomas but unlike the latter they are often arranged in groups.

The clinical picture is often clarified by the distinct histologic findings. The disease primarily affects the sweat glands and this fact suggests the possibility of a relationship of this disorder to a disturbance of the apocrine glands. The epidermis is acanthotic. The sweat glands are dilated. The sweat ducts show a cystic dilatation and their inner surface is lined with small, tongue-like projections. This lining is composed of two or more layers of cells, including an inner columnar and outer cuboidal layer resembling mucinous gland structure. The corium shows a diffuse plasma cell reaction.

These lesions do not undergo malignant transformation. They may be removed by means of excision, electrosurgery or radiotherapy.

10 PSAMMOMA

These rare tumors occur as soft papules or nodules along the vertebral column, located in the deeper layers of the cutis. In a few instances, lesions have been observed on the scalp and are usually not covered by hair. Psammomas are believed to be developmental disturbances since they have been observed in the same regions as meningoceles and cephaloceles. They probably represent displacements of meningeal germ cells. Opinion differs as to whether they are epithelial or endothelial neoplasms.

The diagnosis is invariably a histologic one. The growths are seen in the cutis and subcutis and are composed of linear groups of cells with large vesicular nuclei. Between these linear strands are concentric layers of calcium deposits, supposedly calcified connective tissue. Nerve fibers are also present.

The lesions are benign. Therapy consists of surgical excision.

11 SYRINGOMA (SYRINGOCYSTADENOMA)

This sweat gland adenoma usually affects women in the third decade of their life. The scalp and face are less frequently involved than the trunk. The typical lesion is a soft, smooth papule of a yellowish color, only slightly elevated above the surface of the skin. It resembles multiple benign cystic epithelioma, but, in the latter, the lesions are harder, practically limited to the face, and show pearly elements. In addition, the lesions of multiple benign cystic epithelioma are usually of a flesh or red color, whereas the syringomas are usually of a distinctive yellowish shade. In fact, the yellow color has resulted in confusion with the various types of xanthoma, but the latter are vastly more yellow or orange.

Under the microscope there are no changes of any consequence affecting the epidermis. In the corium, small ropelike or cystlike masses lined with cuboidal epithelium (as in the coil glands) may be seen. These cysts often resemble a drop of water as it is about to separate from the faucet (tear drops) and show a minute tail. This appearance is responsible for their frequent designation as tailed cysts. Inside the cyst there is usually found a pinkish staining substance. There is no surrounding inflammatory reaction. The sections rarely show a very extensive lesion, nor are there too many lesions found in a single field.

These lesions can be removed only by means of destructive measures such as electrosurgery, local caustic applications such as trichloroacetic acid, carbon dioxide snow, electrolysis, excision and radiotherapy.

12 VERRUCA (WART)

Several varieties of wart occur on the scalp. Probably the most common type is the circumscribed, papillary growth with minute, horny, brownish surface projections. A careful examination of the lesions will frequently reveal one or several small satellite verrucae hiding under the adjoining hairs. Their recognition is important inasmuch as a lack of their detection and subsequent removal are usually responsible for a recurrence of the primary growth. Where the lesion is of recent appearance and of rapid growth in an adult, histologic examination should be performed, inasmuch as a rapidly growing squamous cell epithelioma often presents a verrucous appearance on the scalp in its early stages. The scalp is also a not uncommon site for verruca digitata. These warts appear as fingerlike projections with a surmounting horny cap.

Histologically, the epidermis shows a greatly thickened stratum corneum. Atypical areas of parakeratosis may be observed despite retention of a granular layer. Acanthosis is present as a marked downward growth of the rete pegs. Within these pegs the cells are proliferating and mitotic figures may be seen. In the depths where the horn dips down into the rete pegs the granular layer is most pronounced. In these same zones these cells are undergoing a peculiar type of dyskeratosis (vacuolated).

The preferable means of treatment consists of electrodesiccation under local anesthesia. The electrodesiccating needle is first pressed into the skin at the edge of the lesion and remains in the skin until the adjacent tissue is blanched. It is then moved from place to place until the wart is circumvallated. The wart itself is then attacked and entirely dehydrated. The destroyed tissue is removed with a curette down to the soft growing vascular center, which is again dehydrated with the needle of the electrodesiccator.

13 WOOLLY HAIR NEVUS

In 1927 Wise reported two cases of a peculiar form of pilar birthmark previously undescribed. In the first case, portions of the scalp were covered by a dense growth of hair which differed greatly in color, texture, and shape from that of the normal hair covering of the rest of the scalp. The abnormal areas were covered with a uniformly fine golden brown, extremely soft and silky hair which felt "woolly" to the touch. The hair was spiral with undulation of the shaft at 0.5 cm. intervals. The limits of the anomalous growth were sharply defined and no normal hair was found within its borders. While the scalp itself was entirely normal and free from the usual type of birthmarks, the nevoid character of the lesion was indicated by the presence of a pigmented, linear nevus involving the right neck and arm. In the second instance of this anomaly, the entire head from the frontal hair line to the nape of the neck was covered by a heavy growth of light brown, soft, kinky (spiral) or woolly hair. The parietal regions were similarly involved. The patches were sharply demarcated from normal scalp. No nevi, pigmentary changes, or birthmarks were present elsewhere on the body surface. Microscopic examination of the hair from these nevi showed no pathologic changes.

In 1934 Schokking reported a woolly hair mutation in five successive generations of one family. The constant location of the woolly hair on the same portion of the scalp offered a perfect illustration of regular dominant inheritance. The woolly hair in all these cases was abnormally short and thick, i.e., like real wool. Its color varied from dark brown to black in different individuals. Microscopically, it was split and twisted upon its long axis, and the ends were brushlike as in trichorrhexis. Schokking could find no evidence as to how this gene for woolly hair had infiltrated a Dutch peasant family. It appeared most unlikely that it was derived from a racial crossing with a Negro, as the evidence and history of such a crossing were entirely negative, as histories go. (See under Anthropology, page 65, and acquired kinking, page 47.)

II BENIGN CONNECTIVE TISSUE NEW GROWTHS

1 ANGIOMA (VASCULAR NEVUS)

a Port-Wine Stain (Nevus Flammeus)

This lesion is encountered in the scalp in about one third of all newborn infants but sometimes disappears spontaneously. These red discolorations are basically intradermal capillary varicosities, their chief interest being of cosmetic importance. The surface is usually smooth, although it may occasionally be dotted with small nodular tumors or warty elevations. The color is generally a purplish red or violet and is often intensified by crying or coughing. The common location is on the midline just beside the hairy margin in the vicinity of the occipital protuberance although the lesions may extend onto the scalp when they are extensive enough to involve one entire side of the face. These port wine stains usually blanch on pressure.

Histologically, these tumors merely show dilated cutaneous capillaries, lined with endothelium.

The therapy of the port wine stain is notably unsatisfactory. The methods usually recommended include surgical excision if small enough, Grenz ray therapy, applications of thorium X, and solid carbon dioxide. The results with these various methods or treatments are not sufficiently good to warrant the use of any of these modalities. Recently, some of these lesions have been treated by means of tattooing with flesh colored pigment. If a satisfactory color match can be obtained, the results are somewhat satisfactory. Unfortunately, the average person's skin gets darker in the summer and lighter in the winter and the color contrast is not a good one. I have also treated several of these lesions by means of superficial skin removal with sandpaper and with an electric drill. It is too early as yet to evaluate the results in this method of treatment although the dark red lesions show a definite improvement. Many of the lesions in the scalp do not require any form of therapy and if concealment is necessary, there are several satisfactory cosmetic preparations which effectively disguise the lesion.

b Angioma Simplex (Strawberry Mark)

These lesions are generally present at birth. They may be found anywhere on the body and it is not unusual to find one or several on the scalp. They are often

slightly more than pinhead sized at birth, but grow with comparative rapidity on the scalp of infants. This rapidity of growth is an important factor in determining the proper time for treatment of the lesion. If the tumor rapidly enlarges and becomes somewhat lobulated and turgescient with an extreme thinning of the overlying skin, treatment should be administered early. Although a fair percentage of these lesions disappear before the fifth or sixth year, I have also seen a goodly number of them expand to form large, bulky lesions rendering treatment difficult and uncertain with poor cosmetic results. Accordingly, it is my belief that these lesions should be treated shortly after their initial appearance.

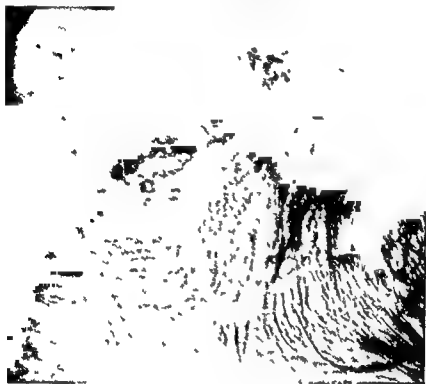


Fig. 290—Angioma simplex of scalp. (Courtesy Clay Adams Co.)

Histologically these lesions show a combination of dilation of pre-existing vessels with proliferation of new channels. The capillary walls are thickened and the vessel spaces are filled with blood and lined with endothelium. Thrombosis may be present in these regions. The surrounding connective tissue shows a moderate degree of proliferation.

c. Cavernous Angioma

These vascular tumors also develop shortly after birth and on the scalp they are often deep-seated, circumscribed lobulated masses blue or bluish black in color. They may even spread to the surrounding tissue involving the cartilage and bone. Some cavernous angiomas are combined with capillary hemangiomas in the over-

lying skin, giving them a purplish red color. They may occur anywhere on the scalp and are usually surmounted by an area of alopecia, although the sides may show a slight growth of vellus hair. These tumors frequently contain one or several blood vessels which may actually be palpated beneath the surface of the growth. This palpation often conveys a feeling that has been likened to that of a mass of worms or of a covered Medusa head. When the deeper structures of the scalp are involved, the subcutaneous portion may be much larger than the superficial surface.

Histologically, the cavernous angiomas show a vascular proliferation which is a combination of dilatation of pre-existing blood vessels and proliferation of new ones. This vascular proliferation may be observed about the sweat glands, hair follicles, and fat globules. There is usually a considerable degree of connective tissue proliferation around these vascular growths. The vessels vary in size but in general are larger than those present in the capillary nevus.



Fig. 291—Cavernous hemangioma. (Courtesy Sutton and Sutton. "Handbook of Diseases of the Skin.")

These lesions are occasionally confused with frontal encephaloceles but the latter are characterized by great distention during coughing and crying and by lobulation and double pulsation. The various types of therapy include repeated applications of solid carbon dioxide, x-ray therapy of the contact type, radium and the injection of sclerosing solutions. These lesions should be treated as soon as possible, depending on the location of the lesion and the rapidity of growth. Prompt treatment is particularly important when the angioma is growing more rapidly than the child inasmuch as such lesions are apt to stretch the skin to such a degree of thinness that secondary infection, hemorrhage and ulceration may result in severe scarring and disfigurement. In addition the endothelial cells lining the vascular space of the tumor are more radiosensitive while they are in their

embryonic stage. As a child grows older, the radiosensitivity of these cells decreases. Beyond the age of 2 years, the effective dosage may be so high as to be dangerous. Every effort should be made, therefore, to begin treatment during the first three months of life. The technique of radium therapy employed in the Johns Hopkins Hospital as reported by Ward and Hendrick is as follows. The amount of radium usually varies between 75 and 200 mg. of elements; the filter is 1 mm. of platinum screened with Monel metal and felt. The distance is 0.5 cm., the time will vary from one to two hours, and the dose is from 180 to 400 mg. hours, depending on the size of the tumor. The average dose = 2 threshold erythemas at 0.5 cm. distance. Radium plaques may be made any size or shape to fit the tumor, permitting the irradiation to be equally distributed. The smaller and more sensitive growths may respond satisfactorily to one application of radium; others require a second, or more rarely a third, spaced at three or four month intervals. Large lesions are treated piecemeal by marking the tumor in segments and treating one area at a time, care being exercised not to overlap the areas. A progressive fading follows for from two to six months. Parents must be kept mindful of this slowness of regression. The number of treatments should be as few as possible to produce the optimal results with a minimum of atrophy, sclerosis, and pigmentation of the skin (the residual effects of overirradiation). The treatment is never repeated until the regression from the previous application has ceased. Too rapid and intensive treatment often produces undesirable scarring and late necrosis. Infants require fewer treatments than older children. In a small number of patients with patches of residual angioma or telangiectasias, applications of solid carbon dioxide suffice to complete the eradication of the lesion. Provided the radium applications to the scalp have not been too intensive, it is surprising to note that normal hair will often regrow through the soft pliable scar that replaces the angioma.

Contact x-ray therapy has also been successfully employed in the treatment of these lesions. The usual technique is similar to that advised by Pendergrass. For the treatment of hemangiomas with the Philips apparatus, Pendergrass using radiation filtered through 2.5 mm. Al gives 200 to 250 r. at intervals of six weeks for a total of four to six doses. This Philips technique gives a total dose of 1,000 to 1,500 r. which compares with a total dose of 2,250 to 3,750 gamma roentgens from the radium applicator. Each dose of 250 r. from the Philips apparatus is equivalent to about three-fourths of an erythema dose, whereas with radium each dose of 675 r. is roughly equivalent to two-thirds of an erythema dose.

According to Andrews, the quality of the radiation from the Philips tube (contact therapy) although it is loosely compared to that produced by radium applicators is different. The radiation from the Philips tube is of a soft quality and even when passed through a filter of 2.5 mm. Al the half value layer according to Quimby is only 1.6 mm. Al. In the treatment of cutaneous diseases, radium plaques so softly filtered are not generally used, but a filter of 0.5 mm. of platinum or 2 mm. of brass is applied. This cuts out all the beta rays and allows 100 per cent gamma radiation. This radiation has a half value layer of about 1.2 cm. of lead.

Pfahler stressed the importance of the hard quality of radiation in the gamma rays of radium for the treatment of hemangiomas. As he states, "Some good results have been obtained and reported by various authors with each kind of radiation—

high voltage and high filtration, low voltage and moderate filtration and contact therapy with very low voltage and little filtration. Pfahler stated that it was his impression that for the treatment of hemangioma the highly filtered radium rays (gamma rays) give the most uniformly good cosmetic results.

Both roentgen and radium rays act by producing an obliterative endarteritis, diffuse sclerosis and fibrous perivascular stroma. The results of the radiation therapy of angiomas depends on two biologic phenomena: the radiosensitivity of the tumor cells and the radiosensitivity of the surrounding stroma. The radiosensitivity of the endothelial cells comprising the blood vessels and the supporting cells is greater, the younger the individual. Rarely, when blood vessels are too large, the angioma does not respond well to radiation. In addition, an associated hyperplasia of fat and connective tissue as evidenced by marked elevation and firmness of the tumors, will also increase the resistance to radiotherapy. This type of lesion may require surgical excision. According to Ward and Hendrick, when there is a combination of capillary and cavernous elements present, the combination of radiation followed by surgery is helpful. The radiation obliterates the capillary growth and makes surgery easier and less radical. Despite these observations, the passage of time finds this author depending less and less on the various radiotherapeutic modalities for the treatment of angioma.

Solid carbon dioxide is of greater value in the treatment of small lesions; there are various techniques employed. It is usually preferable to sketch the lesion roughly through a piece of tracing paper. A piece of dry ice is then shaved down to the approximate size of the sketch. As an additional precautionary measure the lesion may be encircled by several layers of adhesive tape or by pieces of white blotting paper with a central hole approximately the size and shape of the lesion. The carbon dioxide is then applied with moderate pressure for approximately thirty seconds. Temporarily, the area becomes dead white in color and an erythematous reaction appears around the treated site. A bullous lesion subsequently develops on this treated patch and heals by desiccation or rupture leaving an erythematous spot which pales over a period of several months. The entire lesion may be treated by means of a single application or in the larger lesions it is preferable to treat the lesion piecemeal.

Electrosurgical procedures are of value in the treatment of smaller lesions. Ulcerated hemangiomas which have bled repeatedly and profusely require prompt electrodesiccation to stop hemorrhage and destroy the growth. Healing takes place in three to four weeks depending upon the size of the growth. If the hemangioma has been completely destroyed secondary hemorrhage is uncommon. Plastic repair is not often required but if necessary can be planned at a suitable time.

The use of sclerosing solution of the type used in the treatment of varicose veins has also been successful. Cavernous and deeply situated angiomas respond to injections of solutions such as 75 per cent invert sugar, 30 per cent solution of sodium salicylate or the injection of a solution of quinine and ethyl carbonate. Small amounts of sclerosing material are carefully injected into widely separated areas of the lesion. Several injections are given at a single session, but the treatment should not be repeated for one to two weeks. Care must be used in order to prevent a slough which would result in an ugly scar, perhaps requiring surgical excision and a plastic repair.

It should also be mentioned that in the occasional case telangiectasias may be seen on the scalp. One of the common forms is the nevus araneus or spider nevus. It is characterized by a central slightly elevated pinhead sized to slightly larger red dot from which fine blood vessels radiate like the spokes of a wheel. They may occur spontaneously or in association with various systemic and local diseases. They may also occur in association with the syndrome referred to as familial hereditary hemorrhagic telangiectasia (Rendu Osler Weber's syndrome). They are easily destroyed by means of electrolysis.

d Lymphangiomas

These tumors are occasionally seen alone or in combination with blood vessel tumors on the scalp in the form of elevated hairless plaques of different sizes and shapes. They are not as frequent as hemangiomas and may be classified as anomalies of the lymph vessels comparable to those of the blood vessels. They may be roughly divided into capillary and cavernous types, the former involving only the skin, the latter involving the subcutaneous tissue and extending more deeply into the fascia and muscle. Clinically, the tumor appears as a diffuse enlargement of the skin and subcutaneous tissue, moderately compressible and deep blue or purple in color. The lesions referred to as lymphangioma circumscriptum and which appear as small deep seated vesicles generally described as resembling large grains of fish roe are practically never observed on the scalp.

Histologically, the simple lymphangioma consists of overdeveloped lymphatic vessels and spaces lined with epithelium and enveloped in a thin connective tissue base and stroma.

Although these tumors rarely become malignant, they produce large tumors and should be removed when discovered. Surgical excision is the method of choice and it must be thorough in order to prevent the recurrence of the tumor.

2 BLUE NEVUS (JADASSOHN TATCHE)

This blue or slate gray, smooth, round, slightly elevated and firm lesion is usually located on the face or the dorsum of the hands and feet. Not infrequently one may be observed along the scalp margins or in the scalp itself as a solitary lesion. The color is attributed to the location of the melanin pigment in the deep layers of the corium, thereby giving a bluish hue as the color passes through the opaque skin. The diagnosis is not a difficult one inasmuch as the lesion usually appears during infancy or in early childhood and remains as such without increasing in size. This lesion is not infrequently confused with a melanoma when it develops in later life. In fact, the statement is frequently made that all solitary pigmented nevi of this type are malignant and should be dealt with as such. These observations are found throughout the literature and have found their way into the majority of textbooks. It must be recognized that a blue nevus contains cells which are true melanin producing cells of connective tissue origin. These cells are dopa positive and show neither the appearance nor the characteristics of a malignant cell. Malignancy is of infrequent occurrence in the blue nevus.

Histologically these nevi are composed of masses of spindle shaped melanoblasts deep in the corium. These cells have long wavy thin dendritic processes and contain a brown melanin pigment. The pigment may be intra- and extracellular.

In the usual instance the blue nevus does not require treatment. The transition of these lesions from the benign to the malignant state is uncommon. However when located on the scalp they may develop into melanosarcoma as a result of trauma friction or irritation and such cases have been reported. When these lesions do become malignant their growth is slow metastasis is late in developing and superficial ulceration with an inky black discharge may occur. If the patient is an older individual and the lesion is in a site where brushing or combing are of little importance the lesion should be left alone. In younger persons and in those with any history of irritation or of recent change wide and deep surgical excision or electrosurgical removal are the treatments of choice.

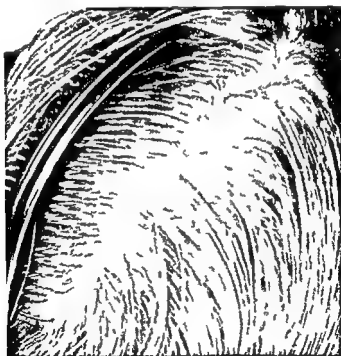


Fig. 29^o—Blue nevus. (Courtesy Dr. Frank Combes.)

3 CICATRIX AND KELOID (SCARS)

Scars are new formations of connective tissue which replace the loss of substance in the corium or fibrous layer of the skin. On the scalp scars usually develop as a result of injury or disease. The size, shape and appearance of the scar is primarily determined by the nature and the extent of the destructive process which preceded its formation. In many instances their contour and configuration are so distinctive on the scalp that a diagnosis of the original condition can be established.

These distinctive features are discussed in greater detail under the headings of the parent disease inasmuch as these patches of scarring or cicatricial alopecia are almost as characteristic as the preceding and more acute symptomatology

A keloid is a dense fibrous growth, usually an overgrowth of a scar, and is characteristically an elevated, smooth surfaced, firm, reddish tumor. They are less common on the scalp than elsewhere on the body. They are most frequently observed on the scalp in the disease entitled folliculitis keloidalis and are discussed under that heading (page 256)

Histologically, these growths are characterized by extremely large, coarse, collagenous fibers, which are interspaced with a few connective tissue cells having small, intensely staining nuclei. Young lesions are fairly vascular and, for that reason, they are more radiosensitive than older keloids. The sweat glands, sebaceous glands, and hair follicles are usually pushed aside by the tumor and frequently exhibit atrophic changes or profuse, small round cell infiltration.

Of the many methods of treatment that have been advocated for these lesions, the most satisfactory are radiotherapy and the application of solid carbon dioxide. Small keloidal scars, less than 2.5 cm in diameter, respond well to applications of carbon dioxide regardless of their age. The usual application, made with either the cryocautery or a solid piece cut to size, consists of a 30 to 60 second exposure with moderate pressure. The area is best left uncovered subsequently although daily applications of an antiseptic dusting powder are of value. It may be necessary to repeat this procedure on one or two subsequent occasions.

X ray or radium therapy is the preferred method of treatment of keloids elsewhere on the body, especially if of recent origin. On the scalp, either method must be utilized with extreme caution, because of the danger of increasing local alopecia. However, if the exposures are limited to the keloid alone, either radiotherapeutic modality is usually a safe and successful one. The author prefers the technique of single exposures to the roentgen rays of 150 r twice weekly (120 kv., 2 mm Al — H V L) for four to six exposures. It is again emphasized that the lesions must be closely shielded.

The greatest problem presented by keloids is the therapy of old lesions, thick globular lesions and recurrent lesions. Although the various radiotherapeutic techniques are preferred by many for the treatment of all types of keloid they are of little value by themselves in these particular variations of scalp keloids. In the majority of these cases radiotherapy must be combined with surgical excision. The surgical procedure should be performed with extreme caution in that the lesion must be excised within its own margins at the extreme edge of the keloid. This is done so as not to stir up any further keloid reaction in the adjacent normal skin. In this type of case, if this technique is not observed, the recurrences that may follow are usually due to the reaction set off in the normal skin by the operation. Closure of the wound should be carried out without tension on the sutures. This is sometimes extremely difficult to accomplish and wide, globular lesions have to be excised piece by piece. This surgical procedure is rarely adequate and must be followed by an adequate course of radiotherapy. In my experience, preoperative radiation is of little value and merely serves to delay wound healing. Postoperative

radiation should be delayed until the wound has healed and barely commenced to show new keloid formation. At this very early stage, the lesions are composed of young fibroblasts and minute, new blood vessels. This tissue is extremely radio-sensitive. The technique employed at this time is similar to that already outlined and should consist in the application of 150 r twice weekly with the factors outlined in the preceding paragraph. If the lesions do not respond completely, the course should be repeated after an interval of two months for a total dosage of 1,200 r. If small areas of keloid are still present, the final treatment should consist in the local application of solid carbon dioxide to the specific areas.

The fully developed scar shows few epidermal changes. The epidermis is thin and composed of but a few layers of cells. The rete cells are absent or few in number, and the rete itself is shallow. In the corium there are dense connective tissue bundles interlacing in all directions. Blood vessels are few and far between in contrast to the early scar where they are usually present in profusion. All glandular appendages including the sweat glands, the sebaceous glands, and hair follicles are absent. The elastic tissue is sparse or entirely absent.

Histologically, the keloid, while it is still young shows a normal epidermis, which thins out later on because of pressure. The corium is separated from the epidermis by a normal layer of connective tissue. In the true keloid, the tumor never begins in the papillae, it begins in the cutis or subcutis. However, the tumor may grow to involve the papillae and, if so, it resembles a false keloid. This differentiation can rarely be made clinically and is of questionable value. In the corium, many cellular elements (fibroblasts) are present. These cells have long oval bodies and large vesicular nuclei. They often occur in whirls and show many mitotic figures. Unlike ordinary fibromas, it is not easy to differentiate the bundles one from the other. As the keloid ages, the connective tissue bundles appear and cellular elements markedly diminish. At this stage the tumor is sharply limited from the surrounding tissue, in the final or end stage of a keloid the tumor is surrounded by a dense capsule of connective tissue. It is for this reason that old keloids are radioresistant.

4 FIBROMAS

These new growths are of connective tissue origin, composed of varying amounts of fibrous tissue cells and fibers developing in the subcutaneous or cutaneous tissue. The lesions may occur as hard nodules and vary in size, shape, and consistency. They may be single or multiple, congenital or acquired and are usually grouped into two general classes on basis of consistency. The soft fibroma (fibroma molle, achrochordon) consists merely of loose, spongy connective tissue. They rarely occur on the scalp, although they may be encountered along the scalp margins at the back of the neck. The hard fibromas (nodular subepidermal fibrosis, fibroma durum) are the result of a productive inflammation associated with fibrosis and are usually associated with trauma. They also are rarely observed on the scalp.

The only fibroma which is encountered on the scalp with any degree of frequency is the neurofibroma. In these instances, it is merely a single manifestation of the disease which is characterized elsewhere by multiple skin tumors frequently

associated with nerve tumors small and large areas of pigmentation in the form of café au lait spots with or without tumor formations frequent evidence of glandular aberration and associated motor skeletal sensory and mental disturbances The hereditary transmission of the disease is a dominant genetic characteristic and the origin of the tumor is considered mesodermal from the connective tissue sheath of nerves (perineurium) On the body, the lesions often number more than one hundred soft molluscous tumors usually pea to egg sized These are not the usual type of lesions encountered on the scalp The lesions on the scalp are usually single, pendulous and large In consistency they are generally velvety soft or semisolid but they may be quite hard The skin covering the tumor is usually devoid of hair soft and pinkish in color They apparently grow from fine cutaneous nerves in the

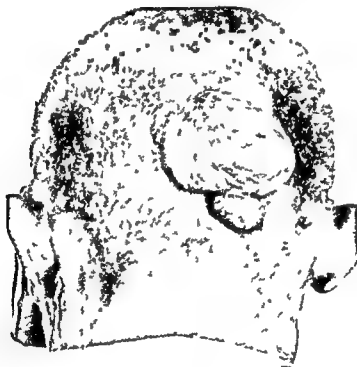


Fig. 91 Neurofibromatosis of von Recklinghausen. This lesion was successfully removed by surgical excision. (Courtesy McCarthy Diseases of the Hair)

skin and frequently follow the distribution of the fifth cranial nerve or of the upper cervical nerves They are situated in the dermis or subcutaneous tissues but they may lie deep in the neck and run upward into the scalp when they arise from nerve trunks or sympathetic nerve These large lesions not infrequently extend to the deeper structures below the skin and may involve adjacent parts of the skull This type of tumor is frequently referred to as a plexiform neuroma or elephantiasis nervorum A large series of these cases were reported by Helmholtz and Cushing They observed that the commonest location is on the scalp and the lesion is likely to begin as a brown spot which eventually becomes soft and slightly pendulous In

the surgical removal of one such lesion, they had great difficulty in controlling the bleeding. When subsequently dissected the removed tissue revealed the neurofibromatous involvement of the auriculotemporal branch of the trigeminal nerve. When the scalp tumor has a dense cordlike base or a cordlike margin extending from it, it is probably derived from and associated with a nerve trunk. This type of tumor is prone to undergo malignant change, and the latter should be suspected if a small, soft tumor gradually develops into a firm, thick, indurated, and dense mass. This malignant transformation is of occasional occurrence and is well under the figure of 30 per cent advanced by some observers. According to Boyd, the incidence of sarcomatous change is only about 8 per cent. It is more apt to appear as a result of an incomplete removal. Sarcomatous change may also be manifested by a change in color from red to an angry purple, the occurrence of ulceration and a more rapid enlargement of the tumor. These tumors are not infrequently observed in the vicinity of the occiput, also a frequent site for lipomas. The latter are less frequently pedunculated and are commonly of a larger size, with a soft 'pillowy' feel, a multilobulated surface, and a median location.

Histologically, the epidermis may appear normal or, if subject to pressure, show atrophic changes. It is usually separated from the underlying tissue by a zone of normal connective tissue. The tumor mass is observed lower down in the corium. It is usually rich in cellular elements and many of these cells show tiny peppery nuclei. The main body of the tumor is composed of fibrous connective tissue cells. These cells are elongated, spindle shaped and fusiform. The nuclei are long and oval. The general impression is that of a fibroma.

Therapy in most instances should be extremely conservative. If the lesion on the scalp is unsightly or frequently traumatized, it should be surgically excised. If the lesion involves a large area, excision must be followed by grafting and plastic repair. Where the tumors are many, there is no procedure of value, and attempted excisions are frequently unsuccessful and often followed by ulceration and sarcomatous change. Patients with extensive neurofibroma should be advised against becoming pregnant inasmuch as this state frequently causes growth and malignant degeneration of these tumors due to the changes in circulating hormonal levels.

4 GRANULOMA PYOGENICUM

This lesion usually appears as a small, pedunculated, reddish tumor of the scalp. It often develops at the site of an injury and is probably the result of an infection. It is considered an exaggeration of proud flesh or exuberant granulation tissue. On the scalp the lesions usually vary in size from 1 mm. to 5 cm. and are reddish or purplish in color. They bleed profusely on the slightest provocation and this often results in the formation of bloody crusts so that the tumor appears almost black instead of reddish. This profuse bleeding following slight trauma often alarms the patient and is usually the symptom responsible for his appearance at the doctor's office. These growths often spring up at the site of some previous abrasion or injury or at the margin of a wound. They are generally painless but may be sensitive to the touch. The surface is often smooth and moist with a shiny,

raspberry like appearance, and for this reason their appearance on the scalp has been confused with that of strawberry nevi, but the history and other clinical features distinguish the two



FIG 294 —Granuloma pyogenicum . . . Usual appearance of lesion at scalp margin (Courtesy Clay Adams Co)



FIG 295 —Granuloma pyogenicum . . . Large tumor clinically suggestive of nevocarcinoma (Courtesy Dr Frank Combes)

Histologically, this tumor of granulation tissue shows a normal epidermis or one that is irregularly acanthotic. In the corium there are many thin walled new and young blood vessels. The stroma between these blood vessels is loose in character, and this aids in differential diagnosis from angiomas in that the stroma of

the latter is adult in character and the blood vessels are neither new nor young. Also, unlike angiomas, there is an infiltration of cellular elements composed mainly of connective tissue cells, mast cells, polymorphonuclear leucocytes, and small round cells.

Therapy depends on the type of lesion encountered. If the granuloma is pedunculated, the tumor may be severed from its base. If this removal is not complete, the lesion will promptly recur. This tendency to relapse is so common a feature that cases of granuloma pyogenicum have been mistaken for sarcoma. It is generally preferable to remove the lesion by means of electrosurgery. Following preliminary anesthesia with Novocain and epinephrine, the latter being of value in controlling bleeding, the exterior of the tissue is either desiccated or coagulated without entering the tumor mass itself. If the lesion rapidly shrinks down to a small, crusted mass, it is easily curetted. Following the removal of this basic crust, the base should be again desiccated in order to insure removal of the entire growth.

6 LIPOMA

These lesions are dermal or subcutaneous new growths composed of fat cells, enclosed within a capsule of connective tissue. The growths are usually lobulated and well encapsulated, but the occasional lesion may be infiltrative in character and send tongue-like projections outward along the blood vessels and between the muscles. They usually vary in size between that of a hazelnut or of an orange, but occasionally grow to a tremendous size. They may occur on the scalp and, in this location, are frequently confused with sebaceous cysts. However, they are more frequently encountered on the back of the neck. Although they are often superficial tumors, they may lie deeply beneath the cervical fascia and in approximately 10 per cent of the cases these deep lying lipomas become malignant. In view of their slow growth and the lack of any accompanying discomfort, the patient frequently does not report for observation until the tumors have attained a considerable size. They may also undergo a more rapid growth following local trauma or the injudicious use of radiotherapy. Subsequent to either change, the lesion may suddenly become painful, sensitive, and hard. This transition may be difficult to detect clinically, but it will be a help to remember that the usual lipoma is definitely an encapsulated tumor, with a soft 'pillowy' feel. In very long standing and large tumors, secondary changes sometimes occur as evidenced by calcification, edema, and liquefaction. When these changes supervene, the overlying skin may necrose and break down discharging an oily putrid material.

Histologically, these lesions are composed of groups of fat cells, slightly larger than normal, and forming small lobules on a supporting capillary network. These lobules are united by strands of connective tissue and enclosed within a connective tissue capsule. According to Montgomery, the lobules are said to 'bear the same relation to the nutrient artery as grapes do to the stem upon which they grow'. If there is a history of rapid growth, many sections should be carefully studied in order to ascertain the presence of malignancy. Malignancy is rare but may consist of a liposarcoma or a myxoliposarcoma. When malignancy supervenes, the microscopic changes consist of islands of embryonic fat tissue along with adult fat and myxom

atous stroma. Scattered in this tissue there are also found extremely large giant cells with foamy cytoplasm. If there is a marked increase in fibrous tissue as well, the probability of malignant change is considerably diminished.

Therapy is usually unnecessary for the smaller growths. When the lesions have enlarged considerably in size, or malignant transformations are suspected, the method of choice is complete extirpation by surgical means. Ward and Hendrick emphasize that tumors deeply located beneath the deep fascia require complete removal of the neoplasm and all of its ramifications from between the muscles and the deep structures. All adherent fascia and muscle must be removed with the tumor.

7 MYXOMA

These tumors are uncommon and do not involve the scalp. However, they may occur along the neck margin and simulate lipomas. They may be differentiated by the fact that they are more deeply situated and more diffuse in outline. When malignancies supervene, the usual growth is a myxosarcoma or a myxoliposarcoma, and the lesions are generally clinically indistinguishable from those observed following malignant transformation of lipomas. At this stage the histologic features and therapeutic approach are similar to that mentioned under the heading of Lipoma (undergoing malignant changes).

8 OSTEOMA

Osteoma is the most common bone tumor of the skull. It has a tendency to occur near the suture lines and more commonly in the frontal bone, although any bone may be involved. It may be single or multiple. Usually the first sign the patient has of an osteoma is the presence of a hard, immovable, painless swelling on the surface of the skull. True bony deposits in the scalp are exceedingly rare, although they have been reported to occur both spontaneously and secondarily following trauma or disease. Clinically, they appear as extremely hard, sharply defined elevated tumors, although a fibrous capsule may soften up the margins. Salzer reported a case in which a coin sized, freely movable, nodule surmounted by an area of alopecia had been present in the scalp for five years. Becker's case had a scalp lesion of fourteen years' duration which occasionally threw off small particles of bone. Taylor and McKenna described a 15 month old child with several bony deposits situated in the extremities scalp and trunk. One of these plaques was well defined hard resilient and elastic. When bent, it would resume its former shape after pressure was removed. The lesions are generally considered to be either misplaced embryonal cells or metaplasia in scarred area, following disease or trauma. Surgical excision of the lesions is the only method of treatment.

III MALIGNANT EPITHELIAL NEW GROWTHS

Malignant epithelial new growths of the scalp are not uncommon. Statistical studies of tumors of the head and neck show the incidence of epithelioma of the scalp to be approximately 5 per cent. These tumors are more apt to occur in individuals with light complexions, blond hair, and blue eyes. If these persons with

thin, dry skins are exposed to sunlight with any degree of frequency, over the years they will develop keratoses of the scalp and subsequent carcinomatous growths. It is also of importance that many benign lesions of the scalp, such as verrucae and nevi that are chronically irritated by combing or brushing the hair, should be adequately removed before malignant transformation develops.

The commonest type of malignancy observed on the scalp is that of a basal cell epithelioma. The individual with greasy, oily, seborrheic skin is much less apt to develop epithelial malignancies of the scalp although such individuals are more susceptible to subsequent epithelial neoplasms over the nose, in the nasolabial folds and on the cheeks. The malignancy is usually squamous cell in type. According to Figge and co workers, this is related in part to a high level of porphyrin excretion. People who excrete a large amount of porphyrin in the sebum are thought to be more susceptible to skin cancer. This observation is offered by these workers as an important etiologic factor in the development of skin cancer about the eyes and nose in individuals with overactive sebaceous glands. Another potential etiologic factor in the development of malignancy of the scalp is a scar resulting from some old trauma or disease. When such scars are present, constant exposure to sunlight, plus frequent scratching and irritation by means of combs and brushes, may stimulate the growth of abnormal cells.

It should be emphasized that, in many instances, carcinoma of the scalp is a preventable disease. Each year, in the various tumor clinics, many individuals are seen with extensive, long standing, and frequently hopeless lesions which should have been recognized and eradicated in their early stages. While many benign growths of the skin elsewhere on the body may persist harmlessly over a period of years, lesions on the scalp are peculiarly prone to stimulation in view of the constant trauma of brushing, combing, and sunlight. Whereas in many instances it is safe to adopt a *laissez faire* policy with lesions elsewhere on the body, this is not the case with tumors of the scalp. It must also be emphasized that when therapy is undertaken, it should be thorough. All lesions, no matter how innocent in appearance, are entitled to histologic examination. The cure of cancer depends upon the administration of adequate therapy, and the latter invariably depends on the reports of histologic study. The ideal method of treatment consists of observation of the lesion, adequate biopsy study, evaluation of the growth, and early, but thorough, treatment. The complete removal of small lesions is of greater importance than the questionable cosmetic result obtained by insufficient treatment. The individual lesion must be studied and the therapy administered which will eradicate it with the first attack or with the first series of treatments.

1 BASAL CELL EPITHELIOMA

The commonest form of tumor affecting the scalp is the basal cell epithelioma. These lesions are comparatively benign in nature, in that they are slow to metastasize and slow to grow. This observation is less applicable to basal cell epithelioma of the scalp, which not infrequently show a high degree of invasiveness. Their origin is from the basal layer of the epidermis or from the accessory structures of the skin. This origin usually is independent of the hair follicles or of the hair matrices.

The clinical appearance is somewhat variable. Probably the most common type is that of a single nodule or a group of elevated, pearly, translucent nodules. These nodules may first appear in an area previously occupied by a keratosis. As these lesions extend and grow, the central portion frequently becomes scaly or crusted and may ulcerate, forming a so called 'rodent ulcer'. In such cases the ulcer is central and superficial with a raised, pearly margin. Another type of basal cell epithelioma is the elevated papillomatous form. These lesions may become extremely large and in appearance resemble large patches of granulation tissue with an elevated slightly nodular margin. At times, there are areas of scarring interspersed with elevated nodules or plaques. It must be recognized that neglected basal cell cancers on the scalp may heal in one area and grow actively in another.



Fig 296—Basal cell epithelioma of the scalp. This lesion illustrates the necessity of histologic study and adequate removal of a new growth of the scalp at the time of the initial treatment. (Courtesy Dr. George Pack.)

The fact that an ulcerated area undergoes subsequent healing following treatment with bland local applications should not lull the therapist into a false sense of security. These lesions should be carefully studied and if further growth occurs they should be eradicated completely, including the scarred areas inasmuch as the latter may contain residual patches of active cells which are usually responsible for subsequent recurrences. Occasionally the scalp may be involved by a morphea-like basal cell carcinoma. These broad and bandlike lesions resemble patches of circumscribed scleroderma in that the surface is smooth, flattened, and ivory colored and of a scarlike nature. However, even in this type the border is characteristically raised, pearly, and wavy.

Histologically the basal cell carcinoma shows a hyperplasia of the basal cell layer. The growth may begin as a small simple bud on the undersurface of the basal layer and grow downward as a band, strand, island, or group of islands. The extent of the invasion is governed by the inflammatory protective zone set up by

the surrounding tissue. This zone is composed of a variable degree of cellular reaction, a protective layer of fibrosis and new granulation tissue. The latter is the least protective of all the layers and is composed of new capillaries and young fibroblasts. The cellular infiltration is characteristically made up of small round cells, plasma cells, and wandering connective tissue cells.

Proper treatment consists of complete eradication of the lesion. The method employed may be electrosurgical, radiotherapeutic, or surgical. A recent method reported by Mohs is also of value as an escharotic method of destruction of skin cancer by a microscopically controlled painstaking method. Mohs removes epithelial skin malignancies layer by layer, checking the sections microscopically until they show freedom from cancer cells.

In a personal communication Mohs discussed a series of twenty five basal cell carcinomas of the scalp. He observed that the degree of invasiveness was high in twenty (80 per cent) and low in the remaining five cases. This tendency for the neoplasm to be rather highly invasive is consistent with the observation that many of these neoplasms of the scalp extend for a considerable distance beyond the clinically detectable margin of the lesion. There are few areas, with the possible exception of the nasolabial fold and the inner canthal region of the eye, where neoplasms are more treacherous. This is due to the frequent presence of "silent" extensions. In this series of twenty five basal cell carcinomas, Mohs observed unexpected peripheral extension in fourteen cases and in five cases there was deep extension in the central portion of the lesion. In six cases the lesions were fairly early and did not exhibit any unusual mode of extension. By far the most frequent mode of extension was peripherally, usually in the deeper part of the subcutaneous connective tissue.

Mohs also had a series of eleven squamous cell carcinomas arising on the scalp. The grade of malignancy was I in three cases, II in four cases, III in four cases and grade IV in no cases. Squamous cell carcinoma showed somewhat less tendency for the peripheral type of spread that was so prominent in basal cell carcinoma, six having central, deep invasion and four having peripheral extensions while one case had both peripheral and central outgrowths. One of the squamous cell carcinomas arose in an old sebaceous cyst.

In the cases in which the neoplasm invaded deeply into the periosteum Mohs found it necessary to remove a layer of bone. It is possible chemosurgically to excise as much bone as appears to be involved as seen by the gross honeycombing. It is difficult to make frozen sections of the bone, and microscopic control is not as easily carried out as it is with the soft tissues. However, if there are soft areas of tissue eroding the bone, it is possible to make frozen sections of that tissue. Otherwise it is necessary to wait some time until decalcification is accomplished. The final layer of fixed bone requires from two to three weeks to form a line of demarcation and it is then possible to remove it in large sheets. The granulation tissue then supports the rapid epithelization of the lesion. Mohs has seen some lesions involving a half or more of the scalp and healing of these large lesions although initially rapid becomes slow and in some cases finally stalls at the later stages. Therefore it is his policy to use split thickness or other types of grafts on the

large scalp lesions as soon as the final layer of fixed bone has been removed and good granulation tissue is present. His results are both esthetic and highly effective.

Electrosurgical and surgical techniques have been described elsewhere. Radiotherapy is of value where the lesions are less than 2.5 cm. in size. The usual techniques are modifications of the original Coutard method. The author administers 600 r every other day for six exposures with dosage factors of 100 kv. and an HVL of 65 mm. Al. Larger lesions may be treated by means of radiotherapy but electrosurgical or surgical removal is preferable. These lesions have frequently infiltrated the entire thickness of the skin and may extend down to the periosteum of the skull. If adequate closure of such wounds is not possible or healing does not seem likely, skin grafting may be necessary.

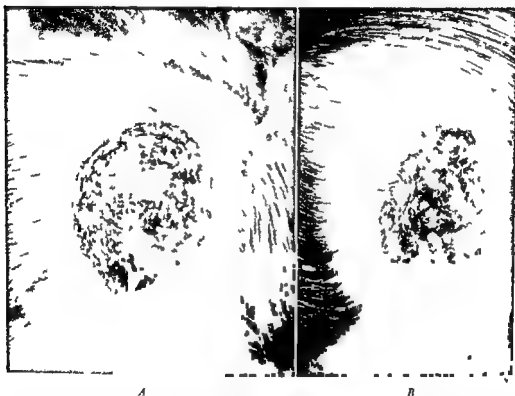
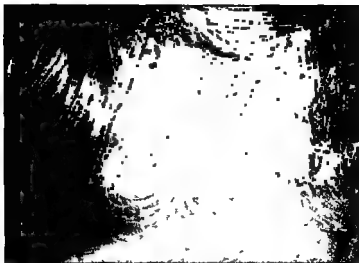


Fig. 297.—Basal cell epithelioma treated chemosurgically. These photographs illustrate a patient with a basal cell carcinoma of 11 years' duration. No previous treatment had been received. The lesion consisted of a partially ulcerated indurated area 3 by 5 cm. on the left anterior scalp (A). The neoplasm was removed in five microscopically controlled stages by the chemosurgical technique of Mohs. The microscopical diagnosis was basal cell carcinoma and there was a definite tendency for the neoplasm to extend in a peripheral direction in the deep subcutaneous connective tissue. On the first day sections were not taken except one for microscopical diagnosis because the carcinoma was grossly visible as a whitish crumbly tissue. However, on the second day no carcinoma was grossly visible and so the area was completely sectioned. Reapplication of the fixative was made only to the areas which proved to be cancerous and on the subsequent days smaller and smaller areas needed treatment until on the fifth day a cancer-free plane was reached. The second photograph of the patient shows the considerable discrepancy between the amount of tissue which had to be removed to eradicate all the cancer and the original lesion as it appeared clinically (B). (Courtesy Dr. Frederic E. Mohs.)



C



D

FIG 297—C and D The third photo shows the healed lesion (C) The patient has remained free of evidence of residual neoplasms for eighteen months The photomicrograph illustrates the carcinoma in the lymphatic plexus in the deep subcutaneous connective tissue (D) (Courtesy Dr Frederic E Mohs)

2 BASAL SQUAMOUS CELL EPITHELIOMA

This type of tumor is clinically indistinguishable from the usual basal cell variety. In fact approximately one out of five epitheliomas diagnosed clinically as a basal cell type will prove histologically to be a basal squamous cell epithelioma. The scalp is one of the common sites of this tumor and the importance in differentiation is merely that this type of lesion may metastasize.

The histologic findings, as the name would indicate, are transitional between those of the basal cell and squamous cell epithelioma.

Therapy is similar to that of the squamous cell epitheliomas.

3 DERMAL APPENDAGE CARCINOMA

Carcinoma of the dermal appendages is uncommon by virtue of the fact that these structures are highly differentiated and their cells are less likely to run wild and revert to immature or malignant forms. The lesions usually arise from hair, sweat glands, or sebaceous glands. Malformed anomalous structures seem to act as initial or primary lesions which, in assuming active growth after prolonged quiescence, become carcinomatous. Squamous cell epitheliomas of the sebaceous glands occasionally appear as subcutaneous or nodular growths or cysts on the scalp and are characterized histologically by the presence of sebaceous cells intermingled with the tumor cells. Loos described 30 cases of sweat gland carcinoma in which the myoepithelial cells necessary for diagnosis were present. According to Montgomery, most lesions described as adenocarcinoma of the sweat glands on careful scrutiny actually prove to be nevus epitheliomatocylindromatosis or syringoma. Setälä has recently reported an excellent study on carcinoma of the dermal appendages. He also investigated the microscopic structure, histogenesis and the degree of malignancy of calcifying epitheliomas of the skin. Many of these lesions were present on the scalp. He suggests that calcifying epitheliomas are derived from hair follicles because of their location, the presence of cuboid or cylindrical cells resembling external hair root sheaths, the presence of squamous cell elements tending to horny degeneration and connective tissue capsule formation with a network of capillaries. Although these lesions are relatively benign, they are locally invasive and show both basal cell and squamous cell transformation. The relative benignity of these lesions may be due to their origin from hair follicles. These studies suggest that if calcifying epitheliomas are derived from hair follicles, some part of the sebaceous apparatus must have the capacity of osteogenesis. If this is not the case, it would be difficult to understand the formation of bone with marrow as described in these cases. These calcifying epitheliomas of the scalp may be differentiated from calcifying epidermal cysts by the basophilic cells in the former and the squamous cells in the latter. Differentiating features of lesser value include the irregular clusterlike arrangement of the basophilic cells in calcifying epithelioma in contrast to the bandlike arrangement of the squamous cells in calcified epidermal cysts and the numerous shadow cells and scarcity of fully keratinized material in epidermal cysts. Lever and Gliksmeyer regard the calcifying epithelioma of Malherbe as a tumor derived from the primary epithelial germ. The basophilic cells



Fig 298 —Squamous cell carcinoma with sebaceous structures, before and after x ray therapy
(Courtesy Dr Kai Setälä)



Fig 299 —Carcinoma with sebaceous and sudoriferous elements, before and after surgical excision. (Courtesy Dr Kai Setälä)

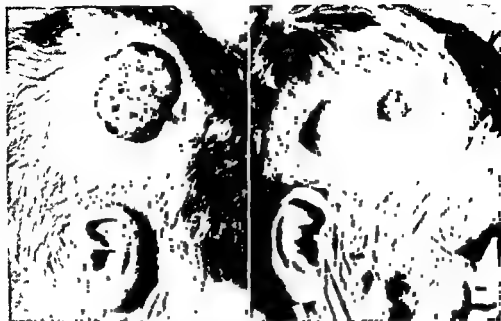


Fig 300—Calcifying epitheliomas of the scalp (type Malherbe) These lesions were adequately treated by surgical excision (Courtesy Dr Kai Setälä)



Fig 301—Squamous cell carcinoma with sebaceous elements Metastases to the regional lymphatics were already present (Courtesy Dr Kai Setälä)



Fig 302 —*A* Low power view of an adnexal carcinoma pilar type proper, showing features characteristic of epithelioma adenoides cysticum. *B* Medium power view of same tumor (Courtesy Drs David Welton, J Elliot and P Kimmelstiel and Arch Dermat & Syph.)



Fig 303 —Transitional type of epithelioma. *A*, Low-power view, showing a stratified squamous cell epithelioma, infiltrating, and forming atypical hair follicles from which glandular structures originate. *B* Medium power view, in which a portion of an atypical hair follicle with tubules in direct continuity with its basal layer may be seen. *C* High power view, showing sudoriparous structures, partially lined by a double layer of cuboidal cells. (Courtesy Drs David Welton, J Elliot, and P Kunmelstiel and Arch Dermat & Syph.)

are primary epithelial germ cells tending to differentiate into keratotic hair cells. Thus, their function is similar to that of hair matrix cells. However, because of their immaturity they cannot produce hairs but only irregular masses of hair cells (shadow cells).

4. METASTATIC CARCINOMA

The scalp is a not infrequent site of cutaneous metastasis from carcinoma elsewhere in the body. The metastasis reaches the scalp by way of the lymphatics or the blood stream. Gates states that in general, skin metastasis has already taken place. Some tumors have a predilection for the scalp, namely about 35 per cent



Fig. 304.—Metastatic carcinoma. The scalp is a not uncommon site of metastases from a carcinoma of the internal organs. This case illustrates hematogenous metastases from a hypernephroma. (Courtesy Sutton and Sutton. Handbook of Diseases of the Skin. Original photograph by Dr. S. Sweitzer.)

of carcinomas of the breast and 20 per cent of all other carcinomas. Montgomery states that in the past too little attention has been paid to the sudden development of solitary or multiple discrete nodules or tumors in the scalp. In a detailed study he observed that the excision and histologic study of nodules which appeared painlessly and asymptotically on the scalp may at times permit the definite diagnosis of metastatic carcinoma even before signs of the primary malignancy have become manifest. Ronchese also described extensive lesions appearing in the scalp as metastases from a primary carcinoma of the prostate gland. In his case, the lesions clinically resembled nevus epitheliomatocylindromatous (turkey tattoo) of the scalp. The histopathologic picture of cutaneous metastases to the scalp is as

general, similar to that of the primary growth. The therapy is, of course, that of the primary growth. Even with extensive metastases, some of these patients have been kept alive by modern methods of hormonal therapy.



Fig. 305.—Metastases to the scalp from a prostatic carcinoma. (Courtesy Sutton and Sutton. *Handbook of Diseases of the Skin*. Original photograph by Dr. T. Ronchese, Arch. Dermat. & Syph.)

5. NEVOCARCINOMA (MELANOTIC OR AMELANOTIC)

This tumor is the most malignant and rapidly fatal of all cutaneous neoplasms. In most instances these lesions develop from a benign pigmented nevus (junction type). Affleck reported a series of cases in which preexisting nevi were found in 84 per cent. In this series, 25 per cent of the entire group involved the scalp, face, neck, and mucous membranes. Pack and associates reported that 23 per cent of all malignant melanomas occurred on the head and neck. Butterworth and Klauder observed that melanomas of the head and neck are especially liable to irritation and are the most likely source of trouble. They state that the most important danger signals are an increase in the size of the lesion or an increase in the degree of pigmentation. In reporting 50 cases and analyzing 598 more from the literature, they found that melanomas had developed from moles located on the head in 16.5 per

cent of their cases. Pack has also reported an interesting series of melanomas in children and refers to them as the prepuberal age group. He states that he has never seen metastasis from a histologically malignant melanoma in a young child. Accordingly, the prognosis in this group is much better than in the more adult group. He states that the microscopic appearance in the prepuberal group is in no way different from the adult group and it is difficult for pathologists to distinguish between them. In Pack's small group of fifteen cases, no metastases occurred. He believes that this tumor has a definite etiologic relationship to circulating hormonal carcinogens originating in the adrenal cortex, pituitary gland, and gonads. He states unequivocally that all solitary, deeply pigmented melanoma found in children should be excised with a good margin of skin around the tumor before the patient reaches the age of puberty. This somewhat radical suggestion is not in complete accord with dermatologic opinion or experience.

The histogenesis of these tumors has given rise to much confusion, and the suggestion that they be called simply melanoma has met with some approval. Nevertheless, melanoma is not a proper name as it signifies "a tumor of pigment" and pigment cannot form a tumor. The proper designation is nevocarcinoma, melanotic or amelanotic. The probability exists that these tumors are neurogenic rather than of epithelial or connective tissue origin, but pathologists differ in their opinions. According to Hertzler, the nevocarcinomas "are born outlaws of the most vicious type and remain so throughout their entire period of existence." According to Ward and Hendrick, clinical observation has permitted these tumors to be divided into two general classifications, that is, cells that are ovoid in character and those that are spindle shaped. The ovoid form is more rapid in its growth and metastasizes early, and is definitely more difficult to eradicate. Spindle cell forms are more likely to remain local, are slow in producing metastasis, and, if metastases are formed, they tend to remain localized for longer periods of time.

On the scalp, these lesions first appear as brown, blue, or black, slightly elevated nevi. Hair may or may not be present in the lesion and is not an indication as to the degree of malignancy or relatively benign state of growth. The statement has frequently been made that lesions containing hair are less apt to be malignant, but this has not been borne out by either clinical or histologic experience. According to Ward and Hendrick, an early sign of malignant change is an enlargement of the primary nevus with subsequent ulceration and frequent bleeding. Other indications include an alteration in color such as darkening, with the subjective symptoms of itching and pain. In other instances there may be little change in the primary lesion but the lymph nodes adjacent to the site show progressive enlargement. In still another group, the first evidence of increased activity was the formation of numerous secondary growths around the primary tumor with or without the enlargement of the draining lymph nodes. This latter form quickly develops widespread metastasis and is most difficult to manage. Although repeated trauma such as following injury or repeated brushing and combing of the scalp, may stimulate growth of the lesion, the question arises as to whether or not this irritation only serves to call the patient's attention to the fact that activity has developed. The first symptom may merely be involvement of a regional group of lymph nodes.

A



B

Fig 306—Nevocarcinoma. *A* Nevocarcinoma of the scalp developing in a nevus superimposed on cutis verticis gyrata. This lesion had already metastasized to the cervical nodes, necessitating a radical neck dissection. *B* Immediate postoperative photograph to show skin grafting. (Courtesy Dr George Pack.)

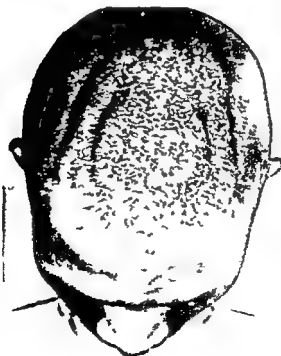


Fig 307 —Cutis verticillata. These examples of a benign lesion are inserted here in order to illustrate the usual benign nature of this nevoid formation in contradistinction to the preceding photograph. A Courtesy Sutton and Sutton. Dances of the Skin. Original photograph by Drs. Kessler and Kessler. B Courtesy Dr. Frank Combes.)

and only by extremely careful examination of the scalp can the small, apparently quiescent nevocarcinoma be located. In such instances, the prognosis is practically hopeless. It is for this reason that all solitary pigmented nevi in areas subject to repeated trauma should be widely excised.

The pathologic changes observed are primarily those of a malignant metaplasia of the ordinary nevus cell. These changes occur often in the junction type of nevus and are usually first observed in the nevus cells at the epidermodermal junction. The nevus cells increase in both size and amount of pigmentation. The cells usually extend to the corium and group themselves in a flasklike arrangement. Mitotic figures are frequent. Although many cells are heavily loaded with pigment, other rapidly growing cells are undifferentiated and fail to form pigment. Pleomorphism is constant. Inasmuch as metastasis is first to the regional lymph nodes and then via the blood stream, early involvement of the lymph and blood vessels may be seen. As the tumor enlarges, all tissue in its path is destroyed.

The treatment of nevocarcinoma is surgical. Many physicians feel that melanomas should be left alone and cite instances of patients who did well without therapy. Unfortunately these instances were rarely confirmed by histologic diagnosis. There is no question of the fact that any pigmented nevus on the scalp subject to frequent trauma or irritation should be removed by wide surgical excision. Radiotherapy has no place in the treatment of melanoma. Recorded observations by Pack and his co-workers show that prior to 1935, the various radiotherapeutic modalities were used by them as the primary mode of treatment. Following 1935 the surgical measures were established as the preferred method of treatment. A comparison of the two methods shows that for a localized melanoma surgery yielded a 38.4 per cent survival after three years as against a 1.6 per cent for irradiation and 17.7 per cent as against none for a five year survival period. If at the time of surgical removal histologic studies show the presence of tumor cells in the lymphatics or blood vessels a poor prognosis can be predicted. Death generally results two to three years after metastasis has occurred, although Broder reported the case of one person who lived ten years after the development of histologically proved cutaneous metastasis and fifteen years after the operative removal of a melanoma of the eye.

Ward and Hendrick state that any solitary melanoma showing growth tendency should be widely excised down through the subcutaneous tissue into the muscles and including at least 1 or 2 cm. of skin around the primary lesion. This kind of extensive removal will often necessitate repair with a split thickness or full thickness of skin graft. Without strict adherence to this surgical principle they state that frequent local recurrences appear in the surrounding skin and metastasis quickly occurs to the local or distant lymph nodes. In addition to a skin margin of 3 cm. the surrounding skin must be undermined for 2 to 3 cm. on all sides and left very thin to prevent the retrograde permeation of the tumor cells through the skin lymphatics, also permitting the removal of a wide border of subcutaneous and cutaneous fat and fascia down to the muscles with the local growth en masse.

The problem of removal of the lymph node bearing area in continuity with the local growth when no nodes are palpable is ever present. The answer depends

upon several factors, namely, the location of the tumor and the age and condition of the patient, along with the findings observed histologically. If there is a history of rapid growth and if the cells histologically are ovoid in character, the tumor should be excised widely as described in continuity with radical dissection of the lymph node bearing area, as developed by Pack.

When there are numerous small lymph glands around the parent tumor or involvement of the nodes in the drainage area, or both, the safest principle is wide excision of the tumor, surrounding skin, subcutaneous skin fascia, and lymph node bearing area en masse. Since melanomas are vicious neoplasms and metastasize early and extensively, Ward and Hendrick believe that all such tumors which microscopically or clinically are suspicious of malignancy should be treated radically. This applies to the more slowly growing spindle cell type as well as to the rapidly spreading ovoid cell variety. This principle pertains to all cases with or without palpable, firm lymph nodes.

Mohs has recently reported on the chemosurgical treatment of melanomas. He states that in a consecutive series of 20 cases of melanoma, successful results were obtained in seven (35 per cent), that primary melanomas apparently were eradicated in 19 of the 20 cases (95 per cent). These results compare favorably with the best results reported in the literature by other workers who used considerably more radical procedures. The systematic microscopic control of excision which characterizes the chemosurgical method is useful in assuring the complete removal of cutaneous melanomas. However, it is necessary to remove an additional zone of tissue after a melanoma free level has been reached because of the danger of satellite deposits in the surrounding lymphatics. The width of this extra zone is varied in proportion to the size of the malignancy of the melanoma. The regional nodes are surgically dissected if enlarged or if the size and degree of malignancy of the primary lesion warrant a prophylactic dissection of nonpalpable nodes.

II SQUAMOUS CELL EPITHELIOMA

Squamous cell epitheliomas are found on the scalp with much less frequency than basal cell epitheliomas. Fundamentally they differ from the basal cell group in that they develop from the prickle cell layer of the skin. The differential points include the more rapid growth and invasion of these tumors, their larger size, and the more rapid development of ulceration. They frequently originate in long standing verrucae, cutaneous horns, epithelial cysts, irritated scars, and keratoses (*s. scaly, senile*). These lesions are not infrequently aggravated by scratching, combing and brushing the hair.

Clinically these lesions usually commence as a scaling, roughened area that gradually becomes indurated and ulcerated. In time there appears a craterlike ulcer with a wide, rolled, pearly border, the latter being wider and more indurated than that of a basal cell epithelioma. The pearly margin is far more characteristic of the basal cell than the squamous cell type. There is often a purplish red areola around the lesion which may extend for some distance. The growth is not infrequently verrucous or warty in character and is often mistaken for a benign lesion on the scalp. A point should be made of the fact that all verrucous lesions on the

scalp of an adult particularly those exhibiting recent growth should be subjected to histologic examination. In addition to these verrucous changes, it is not uncommon to see extensive papillomatous or fungoid cauliflower like elevations. The latter are usually of a lower grade of malignancy than the deeply indurated and ulcerated fast growing tumor. Wilson pointedly emphasized that the degree of malignancy is roughly proportional to the rapidity of ulceration. In the scalp it is not uncommon for a gradually enlarging squamous cell carcinoma to appear merely as a roughened warty patch or small, circumscribed nodule the center of which is frequently ulcerated and is often partially or entirely hidden by the overlying scales and crusts which remain adherent to the lesion. These scales are frequently maintained in place by the surrounding hair. Suspicion should always be aroused by the presence of an indurated base and a hard margin. Gentle curettage of the scales will often reveal the true ulcerative nature of the central region. The growth increases in size by means of peripheral extension undermining the surrounding skin and gradually invading the deeper tissues of the scalp and eventually the periosteum and bony skull as well. Metastases to the adjoining lymph nodes is common and occurs comparatively early in the course of this type of cancer.

Histologically the epitheliomas of the prickly cell type show varying characteristics depending upon their degree of malignancy. They are referred to as intra epidermic if they have not broken through the basal cell margin and extraepidermic if the lesion has pierced that layer. In general the lesions show a dyskeratosis more frequently of the retarded type. In other words the cells do not go on to rapid cornification but rather go backward and become large and more virile with one or several hyperchromatic nucleoli. The cells undergo a marked degree of anaplasia and change both morphologically and physiologically. In other words these cells have completely lost all semblance of restraint and turned wild. Mitotic figures are frequent. A common feature is the so called pearl which is composed of completely cornified epidermal cells. Whorl formation is also frequent and consists of concentrically arranged groups of epithelial cells somewhat resembling the layers of an onion. Broders has divided squamous cell epitheliomas into four grades according to the degree and differentiation of the tumor cells. Attempts at pearl formation, the number of mitoses and evidence of invasion of deeper structures. Other pathologists feel that the tumor is as malignant as its most malignant cell.

Therapy consists in complete eradication of the lesion either by surgical electrosurgical or radiotherapeutic procedures. Radiotherapy may be employed in the treatment of squamous cell epithelioma of the scalp provided the lesion is under 2.5 cm in diameter. A satisfactory technique consists in the application of 900 to 1000 r every other day for a total dosage of 5400 to 10000 r. The dosage factors employed are 100 kv, 20 cm FSD, 2 mm Al filtration with an HVL of 1.8 mm Al. Larger lesions should be treated by means of electrosurgical destruction or cold steel surgery. Many dermatologists prefer the electrocautery or electrocoagulating and electrocutting techniques. Many surgeons prefer radical scalpel surgery. The technique is of itself comparatively unimportant. In the final analysis the cure is effected by the knowledge of the exact depth and area which is to be destroyed and the adequate and complete destruction of the tumor in this area.

7 SUPERFICIAL CARCINOMA (SUPERFICIAL EPITHELIOMATOSIS)

These lesions do not commonly involve the scalp although they may extend into the scalp margins from a patch on the face or neck. Clinically, they are characterized by the development of superficial, sharply circumscribed scaling dermatosis. They are generally observed in the trunk region although they may be seen on the face. The lesions are usually single in number although several or many superficial basal cell carcinomas may appear on one individual. Clinically, the lesions are manifest as red, scaly, dry, irregularly outlined patches. At the border of these patches there is a fine, threadlike pearly elevation. In time, as these lesions progress and enlarge, ulceration and other changes characteristic of basal cell carcinoma may be seen. These lesions are often confused with Bowen's disease or Paget's disease, but the latter are more often exudative, eczematous patches showing a greater acceleration in growth and development and a more inflammatory and subjectively irritating nature. The histologic picture is that of isolated clumps of basal cell tumors closely applied to the undersurface of the epidermis. This multicentric, budlike appearance of the lesion is suggestive of a multicentric origin.

Local measures are of no value, a conclusion which may be reached in the very early stages of this tumor by proper diagnosis and histologic examination. These lesions should be treated by controlled excision, either by surgery or electrosurgery. Where large areas are involved skin grafting may be necessary.

8 XERODERMA PIGMENTOSUM

This rare heredofamilial disease first manifests itself in childhood on the exposed parts of the body (face, upper extremities neck, and temporal regions of the scalp). It is characterized early by brownish, pigmented spots and warty growths. Later, atrophic macules and telangiectases make their appearance, simulating chronic radiodermatitis. The warty lesions usually eventuate in multiple cutaneous epitheliomas often with a final fatal termination. These patients exhibit photosensitivity as a constant symptom and this sensitivity to light may be a causative factor in the progressive development of this disease, as in porphyria. Therapy is limited to avoidance of sunlight and treatment of the individual lesions. The scalp involvement is often an extremely minor component of the over all picture.

IV MALIGNANT CONNECTIVE TISSUE NEOPLASMS

1 SARCOMA CUTIS

There is no uniform agreement as to the classification of cutaneous sarcomas. Sarcomatous changes occur in vascular tumors and they are referred to as angiosarcomas. Changes which occur in individuals with Recklinghausen's disease are referred to as neurofibrosarcomas. Lymphosarcoma is generally classified as a lymphoblastoma. When connective tissue nevi such as the blue nevus become malignant the growth is classified as a melanosisarcoma. Sarcomatous changes also occur in lipomas myomas myxomas fibromas scars and various skin diseases such as lupus vulgaris and erythematosis. True sarcomas of the scalp are not common.

scalp of an adult particularly those exhibiting recent growth should be subjected to histologic examination. In addition to these verrucous changes it is not uncommon to see extensive papillomatous or fungoid cauliflower like elevations. The latter are usually of a lower grade of malignancy than the deeply indurated and ulcerated fast growing tumor. Wilson pointedly emphasized that the degree of malignancy is roughly proportional to the rapidity of ulceration. In the scalp it is not uncommon for a gradually enlarging squamous cell carcinoma to appear merely as a roughened warty patch or small circumscribed nodule, the center of which is frequently ulcerated and is often partially or entirely hidden by the overlying scales and crusts which remain adherent to the lesion. These scales are frequently maintained in place by the surrounding hair. Suspicion should always be aroused by the presence of an indurated base and a hard margin. Gentle curettage of the scales will often reveal the true ulcerative nature of the central region. The growth increases in size by means of peripheral extension, undermining the surrounding skin and gradually invading the deeper tissues of the scalp and eventually the periosteum and bony skull as well. Metastases to the adjoining lymph nodes is common and occurs comparatively early in the course of this type of cancer.

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fibromatous especially in the early fibrous nodules and sarcomatous in the projecting tumors although both phases may intermingle in varying degree in any stage.

Rapid recurrence with advancing rapidity of growth is the rule after their incomplete removal and there is marked resistance to all methods of treatment except complete excision. If this is performed sufficiently early and a considerable zone of the normal tissue surrounding the mass is removed cure usually is obtained.

2 LYMPHOBLASTOMA

This group of diseases is difficult to classify in view of the relatively little knowledge available concerning their specific etiology. Various attempts have been made to divide and subdivide this group according to their benign or malignant predilection, their primary origin from lymph tissues and various other features. However, the close interrelationships of the members of this group result in their imitating not only one another but also the various constitutional diseases and inflammatory processes involving the lymph tissues. An adequate working classification separates the primary lymphomas from the constitutional diseases (leukemias) that also involve the lymph nodes. The primary lymphomas include lymphosarcoma, reticulum cell sarcoma, giant follicle lymphoblastoma, and Hodgkin's disease. The first three conditions are frequently included under the designation of lymphosarcoma. In fact, the lymphosarcomas are often divided into the reticulum or large cell sarcoma and small cell (lymphocytic type) lymphosarcoma and also include sarcoma cutis, giant follicular lymphoblastoma and lymphocytoma. The so-called secondary lymphomas include the constitutional diseases that also involve the lymph tissues. This arbitrary grouping includes the leukemias and granuloma fungoides. It is sometimes difficult to differentiate either clinically or histologically as to whether the lesions are primarily local or constitutional inasmuch as transitions occur from one to the other.

a Hodgkin's Disease

The cutaneous manifestations of this disease resemble those of leukemia cutis in that there are nonspecific types as well as localized and generalized eruptions with the typical histologic features of the disease. The localized forms may appear on the scalp as nodules or tumorlike infiltrations with alopecia over the involved sites. The generalized specific eruption encountered in Hodgkin's disease is that of an erythroderma. The scalp is usually scaly and erythematous with a few scattered patches of remaining hair.

b Leukemia

Three major types of cutaneous lesions occur in leukemia.

1 *Leukemids*.—This group comprises those nonspecific cutaneous manifestations which show none of the characteristic histologic features of leukemia. These lesions involve the skin primarily although the scalp may be affected. They appear during the course of leukemia and are clinically indistinguishable from those occurring during the course of Hodgkin's disease and other lymphoblastomas as well as the hemorrhagic diatheses, prurigo and other disorders of the integument.

Other signs and symptoms include pruritus erythema multiforme diffuse exfoliative dermatitis purpura eczematous dermatitis herpes zoster superficial ulceration ulcerative gingivitis and bleeding of the gums

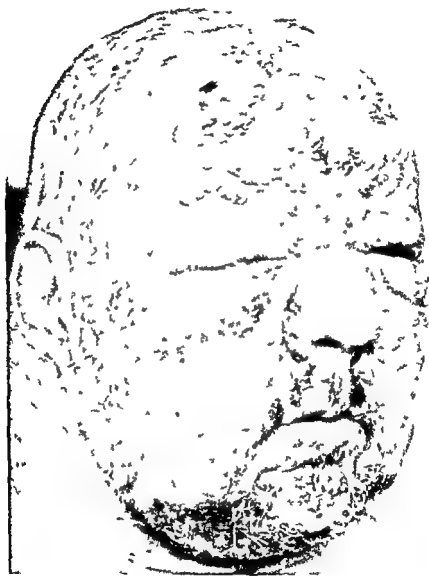


Fig. 11. Leukemia. (Courtesy Sutton and Sutton, *Diseases of the Skin*. Original photograph by Dr. G. W. Wende.)

2. **Lymphadenosis Cutis Circumscripta**—These true leukemic infiltrations of the skin usually appear as discrete doughy nodules or tumors of a bluish red color during the course of lymphatic leukemia. They may also occur as nodular subcutaneous infiltration covered by normal skin or as superficial macules. The face is the most common site although the extensor aspects of the extremities (especially

the backs of the hands) are also frequently involved. The tumors may be round surfaced or flat topped elevations varying from 1 to 8 cm in size. The scalp is occasionally involved by a nodule or a tumorlike area of alopecia and infiltration. Ulceration and scarring rarely occur, and involution (either spontaneous or radiotherapeutic) leaves no trace of the lesions. These tumors have been observed in all forms of leukemia but are far more frequent in the chronic lymphatic type.

3 Lymphadenosis Cutis Universalis—This generalized erythrodermic infiltration of the skin with microscopically true leukemic cells is characterized by a universal symmetrical swelling and redness of the integument. This exfoliative dermatitis occurs most often in lymphatic leukemia. The thickening of the skin



Fig 309—Myeloid leukemia involving the scalp (Courtesy Sutton and Sutton, *Diseases of the Skin*. Original photograph by Drs. Ketron and Gay.)

accentuates all the body folds and presages the development of a leonine countenance. The skin is usually light or dark red in color, scaly, and intensely itchy. In monocytic leukemia, which is accompanied by skin lesions in 50 per cent of the cases, exfoliative erythroderma is a common finding. As in all forms of erythroderma, regardless of origin, the scalp becomes erythematous, scaly, and often crusted. The hair usually falls out in a diffuse spread and may progress to a total alopecia.

■ Giant Follicular Lymphoblastoma

This disease is a forerunner of the more serious types of lymphoblastoma. It commences as a massive splenomegaly accompanied by progressive painless lymphadenopathy.

adenopathy. It is not infrequently associated with a generalized erythroderma in which the scalp participates with erythema, scaling, and diffuse alopecia. The disease is analogous to the herald patch of pityriasis rosea in that it is a cutaneous heraldic portent of the more serious forms of lymphoblastoma.



FIG. 310.—Erythroderma tumors and alopecia in mycosis fungoides. (Courtesy Dr. Frank Combes.)

d. Mycosis Fungoides

This severe and usually fatal disorder is characterized in its early stages by a pruritic nonspecific erythematous eruption of the skin and scalp. In time, infiltrated plaques and nodules supplement this eruption, and on the face the clinical appearance is often similar to that of leprosy (leonine facies). The eyelashes and eyebrows often fall out and the scalp shows a partial or complete diffuse alopecia in addition to the presence of erythematous infiltrated plaques of actual nodules.



Fig 311 —Plaque alopecia in association with lesions of mycosis fungoides (Courtesy Dr Marion Sultzberger)



Fig 312 —Involvement of scalp in demodic type of mycosis fungoides (Courtesy Dr A Boghosian)

and tumors In the final stage, the tumors on the scalp share the characteristic fungoid "rotten tomato" ulcerative appearance of the lesions present elsewhere on the body

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APPENDIX

FORMULARY

SHAMPOOS

- | | | | | |
|-----------------------------|---------|-------------------------------|------------------|--|
| (1) (Savill) | | (10) | Pine Tar Shampoo | |
| Soft soap | | Soluble pine tar liquid | 40 cc | |
| Oil of cade | | Soft soap | 320 Gm | |
| Alcohol of each equal parts | | Alcohol | 500 cc | |
| Perfume q s | | Water q s ad | 1000 cc | |
| (2) | | (11) (de Navarre) | | |
| Thymol | 08 Gm | Cocornut fatty acids | 150 cc | |
| Oil of cade | 16 cc | Castor fatty acids | 40 cc | |
| Sodium baborate | 16 Gm | Potassium hydroxide 100% | 40 Gm | |
| Sodium carbonate | 04 Gm | Triethanolamine | 30 cc | |
| Tr green soap q s ad | 1000 cc | Water | 740 cc | |
| (3) (Goodman) | | (12) (Thomssen) | | |
| Tr of soap bark | 20 cc | Almond Oil Shampoo | | |
| Borax | 100 Gm | Almond oil | 50 cc | |
| Bay Rum | 20 cc | Palm kernel oil | 130 cc | |
| Water q s ad | 1000 cc | Sesame oil | 120 cc | |
| (4) (Goodman) | | Caustic potash 85% | 515 Gm | |
| Olive Oil Jelly Shampoo | | Caustic soda 95% | 10 Gm | |
| Castile Soap shavings | 400 Gm | Alcohol | 50 cc | |
| Olive oil | 40 cc | Water | 5785 cc | |
| Soft soap | 40 Gm | Perfume | 10 cc | |
| 5% potass carb sol | 40 cc | (13) | | |
| Alcohol (50%) q s ad | 1000 cc | Liquid Shampoo | | |
| (5) | | Cocornut oil | 60 cc | |
| Oily Scrlp Shampoo | | Cottonseed oil | 80 cc | |
| Soft soap | 600 Gm | Stearic acid | 40 Gm | |
| Water | 200 cc | Potassium carbonate | 10 Gm | |
| Alcohol q s ad | 1000 cc | Potassium hydroxide | 40 Gm | |
| (6) | | Alcohol | 40 cc | |
| Dry Shampoo | | Talcum | 10 Gm | |
| Starch | | Water q s ad | 1000 cc | |
| Sodium bicarbonate of each | | (14) (Goodman) | | |
| equal parts | | Oleic acid | 240 cc | |
| Perfume q s | | Liquid petrolatum | 240 cc | |
| (7) | | Sulfonated castor oil | 240 cc | |
| Trisodium phosphate | 40 Gm | Sodium bicarbonate | 10 Gm | |
| Silica gel | 200 Gm | Alcohol q s ad | 1000 cc | |
| Ppt chalk q s ad | 1000 Gm | (15) | | |
| (8) | | Ethereal Soap Solution | | |
| Shampoo Powder | | Oleic acid | 200 cc | |
| Potassium carbonate | 40 Gm | Potassium hydroxide | | |
| Powdered soap q s ad | 1000 Gm | (sat sol) | 50 cc | |
| (9) | | Alcohol | 120 cc | |
| Ether Soap | | Perfume | 20 cc | |
| Ether | 120 cc | Fther q s ad | 1000 cc | |
| Stronger ammonia water | 06 cc | (16) | | |
| Soft soap q s ad | 1000 Gm | Scalp Milk | | |
| | | Stearic acid | 60 Gm | |
| | | Liquid petrolatum | 320 cc | |
| | | Triethanolamine | 20 cc | |
| | | Water q s ad | 1000 cc | |

- (17)
- | | |
|-----------------|----------|
| Olive oil | 240 cc |
| Cocoonut oil | 240 cc |
| Triethanolamine | 20.40 cc |
| Carbitol q s ad | 1000 cc |
- (18) (Thomssen)
- Olive Oil Shampoo
- | | |
|--------------------|----------|
| Olive oil | 96 cc |
| Palm kernel oil | 54 cc |
| Cocoonut oil | 80 cc |
| Caustic potash 85% | 4.46 Gm |
| Caustic soda 95% | 11 Gm |
| Alcohol | 28 cc |
| Water | 67.64 cc |
| Perfume | 10 cc |
- (19) (Harry)
- Triethanolamine lauryl sulfate
- | | |
|--------------------------------|--------|
| Triethanolamine lauryl sulfate | 400 cc |
| Water | 600 cc |
| Color | q s |
| Perfume | q s |
- (20) (Harry)
- Soapless Shampoo
- | | |
|-----------------------|--------|
| Sulfonated olive oil | 160 cc |
| Sulfonated castor oil | 160 cc |
| Water | 680 cc |
- (21) (de Navarre)
- | | |
|-----------------------------|--------|
| Sulfonated olive oil (90%) | 250 cc |
| Sulfonated castor oil (90%) | 710 cc |
| Color | q s |
| Perfume | q s |
| Preservative | q s |
- (22)
- Soapless Shampoo
- | | |
|-----------------------------|---------|
| Sodium alkyl aryl sulfonate | 200 Gm |
| Glycerin | 80 cc |
| Water q s ad | 1000 cc |
| Perfume | q s |
| Color | q s |
- (4)
- | | |
|----------------|---------|
| Salicylic acid | 10 Gm |
| Resorcin | 20 Gm |
| Boric acid | 50 Gm |
| Glycerin | 60 cc |
| Alcohol | 500 cc |
| Water q s ad | 1000 cc |
- (5)
- | | |
|-----------------|---------|
| Chloral hydrate | 10 Gm |
| Glycerin | 60 cc |
| Alcohol | 320 cc |
| Bay rum q s ad | 1000 cc |
- (6)
- | | |
|-------------------|---------|
| Resorcin | 10 Gm |
| Chloral hydrate | 06 Gm |
| Camphor | 06 Gm |
| Tr of cantharides | 12 cc |
| Tr of capsicum | 12 cc |
| Glycerin | 60 cc |
| Alcohol | 320 cc |
| Water q s ad | 1000 cc |
- (7)
- | | |
|------------------------|---------|
| Resorcinol monoacetate | 25 cc |
| Spirit of formic acid | 20 cc |
| Tr of cantharides | 20 cc |
| Betanaphthol | 10 Gm |
| Alcohol | 640 cc |
| Water q s ad | 1000 cc |
- (8)
- | | |
|------------------------|---------|
| Resorcinol monoacetate | 10 cc |
| Menthol | 20 Gm |
| Alcohol | 500 cc |
| Glycerin | 40 cc |
| Water q s ad | 1000 cc |
| Perfume q s | |
- (9) (Thomssen)
- | | |
|------------------------|----------|
| Resorcinol monoacetate | 40 cc |
| Mercuric chloride | 0.12 Gm |
| Spirit of formic acid | 150 cc |
| Castor oil | 20 cc |
| Alcohol | 78.83 cc |
| Perfume | 0.05 cc |
- (10)
- | | |
|-----------------|---------|
| Tannic acid | 10 Gm |
| Chloral hydrate | 10 Gm |
| Castor oil | 10 cc |
| Soy bean oil | 60 cc |
| Witch hazel | 200 cc |
| Alcohol q s ad | 1000 cc |
- (11) (Goodman)
- Alkaline Wash
- | | |
|---------------------------|---------|
| Stronger ammonia water | 160 cc |
| Almond oil | 160 cc |
| Tr of cantharides | 20 cc |
| Oil of orange | 10 cc |
| Spirit of rosemary q s ad | 1000 cc |
- (12) (Goodman)
- | | |
|-----------------------|------------|
| Sulfonated castor oil | 200.400 cc |
| Sulfonated olive oil | 60.100 cc |
| D ethylene glycol | 10 cc |
| Triethanolamine | 10 cc |
| Water q s ad | 1000 cc |

HAIR LOTIONS

(Mildly keratolytic and Rubefacient)

- 2 (1)
- | | |
|----------------------|---------|
| Cholesterol | 0.4 Gm |
| Lecithin | 0.1 Gm |
| Carbon tetrachloride | 40 cc |
| Glycerin | 60 cc |
| Water q s ad | 1000 cc |
- (2)
- | | |
|---------------------------|---------|
| Tr of capsicum | 12 cc |
| Tr of cantharides | 12 cc |
| Pilocarpine hydrochloride | 12 Gm |
| Stronger ammonia water | 40 cc |
| Alcohol | 100 cc |
| Water q s ad | 1000 cc |
- (3)
- | | |
|------------------------------|---------|
| Oxyquinoline sulfate | 10 Gm |
| Water | 320 cc |
| Sulfonated castor oil q s ad | 1000 cc |

- (13) **Scalp Lotion for Dark Hair**
 Resorcin 18 Gm
 Salicylic acid 18 Gm
 Mercury bichloride 0.1 Gm
 Glycerin 10 cc
 Cologne water 200 cc
 Alcohol 300 cc
 Water q s ad 1000 cc
- (14) **Scalp Lotion for Blond Hair**
 Chloral hydrate 18 Gm
 Salicylic acid 18 Gm
 Mercury bichloride 0.1 Gm
 Glycerin 10 cc
 Spirit of lavender 50 cc
 Alcohol 450 cc
 Water q s ad 1000 cc
- (15) (Thomssen)
 Alcohol 200 cc
 Bay rum 400 cc
 Camphor 0.5 Gm
 Menthol 0.3 Gm
 Tr of Quillaja 100 cc
 Water 29.2 cc
- (16)
 Quinine hydrochloride 0.5 Gm
 Chloroform 0.5 cc
 Alcohol 200 cc
 Glycerin 15 cc
 Bay rum 250 cc
 Tr of cudbear 30 cc
 Rose water q s ad 1000 cc
- (17)
 Sage Lotion
 Oil of sage 0.15 cc
 Tr of cantharides 12 cc
 Glycerin 30 cc
 Menthol 15 Gm
 Bay rum 125 cc
 Oil of bergamot 15 cc
 Alcohol 250 cc
 Water q s ad 1000 cc
- (4) (Sir Norman Walker)
 Lactic acid 15 cc
 Oleoresin of capsicum 25 Gm
 Bay rum q s ad 1000 cc
 (Note Apply twice a day, cautiously at first, then with more and more vigor as scalp gets used to it)
- (5) (Sabouraud)
 Sulfur 20 Gm
 ~~~~~ 200 Gm  
 Wash scalp in the morning and brush vigorously)
- (6) (Max Joseph)  
 Tr of cantharides 30 cc  
 Alcohol (50%) 1000 cc
- (7)  
 Tr of cantharides 125 cc  
 Camphor 125 Gm  
 Alcohol 125 cc  
 Distilled water q s ad 1000 cc
- (8)  
 Precipitated sulfur 10 Gm  
 Alcohol 200 cc  
 Distilled water q s ad 1000 cc
- (9) Chloral hydrate 20 100 Gm  
 Bay rum 1000 cc
- (10)  
 Resorcin 32 Gm  
 Betanaphthol 0.12 0.8 Gm  
 Mercury bichloride 0.05 Gm  
 Alcohol 220 cc  
 Distilled water q s ad 1000 cc  
 (Note For dark hair only)
- (11) (Mackee)  
 Oleoresin of capsicum 0.01 0.12 Gm  
 Croton oil liniment 0.8 cc  
 Quinine hydrochloride 0.4 Gm  
 Mercury bichloride 0.05 Gm  
 Tr of cantharides 3.2 64 cc  
 Alcohol 225 cc  
 Distilled water q s ad 1000 cc  
 (Note Will not discolor light hair  
 Sodium sulfocarbolate 16 Gm can be used in place of the mercury bichloride)

**HAIR LOTIONS**(Moderately Keratolytic and Rubefacient  
Occasionally Irritating and Sensitizing)

- (1) (Sabouraud)  
 Potassium nitrate 0.16 Gm  
 Distilled water 100 cc  
 Alcohol (90%) q s ad 1000 cc
- (2) (Savill)  
 Carbolic acid 10 Gm  
 Tr of cantharides 40 cc  
 Alcohol 320 cc  
 Bay rum 80 cc  
 Distilled water q s ad 1000 cc
- (3)  
 Tr of jaborandi 60 cc  
 Tr of cantharides 120 cc  
 Salicylic acid 20 Gm  
 Glycerin 60 cc  
 Alcohol q s ad 1000 cc
- (12) (Bulkley)  
 Quinine sulfate 0.75 Gm  
 Zinc sulfate 0.4 Gm  
 Tr of cantharides 75 cc  
 Alcohol 90 cc  
 Glycerin 90 cc  
 Bay rum q s ad 1000 cc
- (13)  
 Chloral hydrate 16 Gm  
 Salicylic acid 16 Gm  
 Castor oil 30 cc  
 Cologne water 500 cc  
 Alcohol (50%) q s ad 1000 cc

- |                            |          |                            |           |
|----------------------------|----------|----------------------------|-----------|
| (14) (Goodman)             |          | Tr of capsicum             | 10 cc     |
| Coal tar solution          | 40 cc    | Castor oil                 | 40 cc     |
| Castor oil                 | 40 cc    | Alcohol                    | 750 cc    |
| Alcohol                    | 620 cc   | Distilled water q s ad     | 1000 cc   |
| Distilled water q s ad     | 1000 cc  |                            |           |
| (15) Sodium undecylenate   | 0.5 Gm   | (24) Salicylic acid        | 10.30 Gm  |
| Salicylic acid             | 30 Gm    | Resorcin                   | 10.30 Gm  |
| Oxyquinoline sulfate       | 10 Gm    | Castor oil                 | 40.100 cc |
| Alcohol                    | 600 cc   | Cologne water q s ad       | 1000 Gm   |
| Distilled water q s ad     | 1000 cc  |                            |           |
| (16) (Goodman)             |          | (25) (Goodman)             |           |
| Chloral hydrate            | 20 Gm    | Resorcin                   | 10 Gm     |
| Tannic acid                | 10 Gm    | Rectified oil of tar       | 10 cc     |
| Castor oil                 | 40 cc    | Soft soap                  | 40 Gm     |
| Soybean oil                | 100 cc   | Alcohol                    | 120 cc    |
| Witch hazel extract        | 320 cc   | Distilled water q s ad     | 1000 cc   |
| Alcohol q s ad             | 1000 cc  |                            |           |
| (17) (Goodman)             |          | (26) Quinine hydrochloride | 10 Gm     |
| Anthrasol                  | 20 Gm    | Chloral hydrate            | 20 Gm     |
| Glycerin                   | 40 cc    | Castor oil                 | 40 cc     |
| Oil of neroli              | 02 cc    | Alcohol                    | 500 cc    |
| Tr of green soap           | 200 cc   | Cologne water q s ad       | 1000 cc   |
| Alcohol q s ad             | 1000 cc  |                            |           |
| (18) Salicylic acid        | 0.5 Gm   | (27) (Goodman)             |           |
| Resorcinol monoacetate     | 10.40 cc | Quinine hydrochloride      | 06 Gm     |
| Castor oil                 | 40 cc    | Tr of cantharides          | 12 cc     |
| Spirit of lavender         | 200 cc   | Balsam of Peru             | 20 Gm     |
| Oil of bergamot            | 04 cc    | Distilled water            | 120 cc    |
| Alcohol q s ad             | 1000 cc  | Alcohol q s ad             | 1000 cc   |
| (19) Chloral hydrate       | 15 Gm    | (28)                       |           |
| Tr of capsicum             | 25 cc    | Hexachlorophene            | 10 Gm     |
| Resorcinol monoacetate     | 20 cc    | Menthol                    | 02 Gm     |
| Castor oil                 | 100 cc   | Chloral hydrate            | 05 Gm     |
| Tr of cantharides          | 20 cc    | Glycerin                   | 150 cc    |
| Spirits of lavender q s ad | 1000 cc  | Cologne water              | 200 cc    |
|                            |          | Distilled water q s ad     | 1000 cc   |
| (20) (Goodman)             |          | (29) (Goodman)             |           |
| Salicylic acid             | 06 Gm    | Antiseptic Scalp Lotion    |           |
| Chloral hydrate            | 06 Gm    | Oxyquinoline sulfate       | 10 Gm     |
| Acetone                    | 10 cc    | Isopropyl alcohol          | 400 cc    |
| Glycerin                   | 60 cc    | Distilled water q s ad     | 1000 cc   |
| Alcohol                    | 240 cc   |                            |           |
| Distilled water q s ad     | 1000 cc  | (30)                       |           |
| (21)                       |          | Salicylic acid             | 10 Gm     |
| Resorcin                   | 10.20 Gm | Resorcin                   | 05 Gm     |
| Salicylic acid             | 10.20 Gm | Chloral hydrate            | 20 Gm     |
| Castor oil                 | 50 cc    | Alcohol                    | 600 cc    |
| Alcohol                    | 500 cc   | Distilled water q s ad     | 1000 cc   |
| Cologne water q s ad       | 1000 cc  |                            |           |
| (22) (Goodman)             |          | (31)                       |           |
| Resorcin                   | 10 Gm    | Balsam of Peru             | 10 Gm     |
| Betanaphthol               | 20 Gm    | Tr of cantharides          | 20 cc     |
| Chloral hydrate            | 10 Gm    | Cologne water              | 600 cc    |
| Sulfonated castor oil      | 240 cc   | Oil of lavender            | 10 cc     |
| Alcohol q s ad             | 1000 cc  | Distilled water q s ad     | 1000 cc   |
| (23)                       |          | (32)                       |           |
| Resorcin monoacetate       | 10.30 cc | Salicylic acid             | 05 Gm     |
| Sodium undecylenate        | 10 Gm    | Tr of capsicum             | 05 cc     |
| Chloral hydrate            | 15 Gm    | Tr of cantharides          | 05 cc     |
|                            |          | Hexachlorophene            | 05 Gm     |
|                            |          | Bay rum                    | 600 cc    |
|                            |          | Distilled water q s ad     | 1000 cc   |

- (33) (Goodman)
- |                |         |
|----------------|---------|
| Cholesterin    | 10 Gm   |
| Lecithin       | 0.4 Gm  |
| Castor oil     | 40 cc   |
| Alcohol q s ad | 1000 cc |
- (34) (Goodman)
- |                      |         |
|----------------------|---------|
| Cholesterin          | 0.4 Gm  |
| Carbon tetrachloride | 40 cc   |
| Glycerin             | 40 cc   |
| Orange flower water  | 400 cc  |
| Alcohol q s ad       | 1000 cc |
- (35) (Goodman)
- |                        |         |
|------------------------|---------|
| Cholesterin            | 10 Gm   |
| Liquid petrolatum      | 60 cc   |
| Sodium choleate        | 0.4 Gm  |
| Glycerin               | 60 cc   |
| Borax                  | 10 Gm   |
| Distilled water q s ad | 1000 cc |
- (36)
- |                           |         |
|---------------------------|---------|
| Pilocarpine hydrochloride | 10 Gm   |
| Spirit of lavender        | 200 cc  |
| Resorcin                  | 10 Gm   |
| Castor oil                | 40 cc   |
| Alcohol                   | 600 cc  |
| Water q s ad              | 1000 cc |
- (37)
- |                           |          |
|---------------------------|----------|
| Resorcin                  | 10.30 Gm |
| Castor oil                | 50 cc    |
| Pilocarpine hydrochloride | 20 Gm    |
| Tr of cantharides         | 50 cc    |
| Alcohol (85%) q s ad      | 1000 cc  |
- (38) (Goodman)
- |                           |         |
|---------------------------|---------|
| Pilocarpine hydrochloride | 0.6 Gm  |
| Quinine bisulfate         | 10 Gm   |
| Betanaphthol              | 40 Gm   |
| Castor oil                | 80 cc   |
| Alcohol                   | 500 cc  |
| Distilled water q s ad    | 1000 cc |
- (39) (Goodman)
- |                        |         |
|------------------------|---------|
| Capsicin               | 0.12 Gm |
| Zinc sulfocarbolate    | 10 Gm   |
| Tr of cantharides      | 80 cc   |
| Spirit of formic acid  | 80 cc   |
| Alcohol                | 640 cc  |
| Distilled water q s ad | 1000 cc |
- (40)
- |                |          |
|----------------|----------|
| Balsam of Peru | 0.5 Gm   |
| Salicylic acid | 10.50 Gm |
| Castor oil     | 50 cc    |
| Cologne water  | 300 cc   |
| Alcohol q s ad | 1000 cc  |
- (41)
- |                           |          |
|---------------------------|----------|
| Pilocarpine hydrochloride | 0.5 Gm   |
| Salicylic acid            | 10 Gm    |
| Resorcinol monoacetate    | 10.50 cc |
| Cologne water             | 400 cc   |
| Alcohol q s ad            | 1000 cc  |
- (42) (Andrews)
- Very Drying Lotion for Excessively Oily Scalp
- |                  |          |
|------------------|----------|
| Formaldehyde 40% | 16.40 cc |
|------------------|----------|
- Sulfuric acid dilute 60 cc.
- Alcohol 32.64 cc
- Acetone 60.240 cc
- Distilled water q s ad 1000 cc
- Oil of rose geranium 0.3 cc
- (43) (Andrews)
- |                                                                                                                                         |          |
|-----------------------------------------------------------------------------------------------------------------------------------------|----------|
| Zinc sulfocarbolate                                                                                                                     | 16.32 Gm |
| Resorcin                                                                                                                                | 16.64 Gm |
| Betanaphthol                                                                                                                            | 0.8 Gm   |
| Alcohol                                                                                                                                 | 500 cc   |
| Distilled water q s ad                                                                                                                  | 1000 cc  |
| Perfume with oil of violet or oil of bay                                                                                                |          |
| Color with Tr Perssonis or Bismarck brown                                                                                               |          |
| (Note For dark hair only Violet odor and color for women brown color and bay odor for men Oleo resin of capsicum 16.48 cc may be added) |          |
- (44) (Harry)
- |                |        |
|----------------|--------|
| Resorcin       | 50 Gm  |
| Tr of capsicum | 50 cc  |
| Castor oil     | 50 cc  |
| Alcohol (90%)  | 850 cc |
| Perfume        | q s    |
- (45) (Harry)
- |                   |        |
|-------------------|--------|
| Isopropyl alcohol | 500 cc |
| Distilled water   | 496 cc |
| Betanaphthol      | 0.2 Gm |
| Salicylic acid    | 0.2 Gm |
| Perfume           | q s    |
- (46) (de Navarre)
- |                                    |         |
|------------------------------------|---------|
| Resorcinol monoacetate             | 2.5 cc  |
| Unsaturates (methyl linoleate 50%) | 2.5 cc  |
| Oil of cloves                      | 0.5 cc  |
| Cinnamon                           | 20 Gm   |
| Alcohol D q s ad                   | 1000 cc |
- (47) (de Navarre)
- |                                   |         |
|-----------------------------------|---------|
| Cologne water                     | 100 cc  |
| Tr of cinchona                    | 100 cc  |
| Diethylene glycol monoethyl ether | 50 cc   |
| Tr of quillaja                    | 50 cc   |
| Orange flower water               | 100 cc  |
| Alcohol S D q s ad                | 1000 cc |
- (48) (de Navarre)
- |                                |         |
|--------------------------------|---------|
| Cholesterol                    | 0.5 Gm  |
| Soybean lecithin               | 0.15 Gm |
| Propylene glycol               | 50 cc   |
| Isopropyl alcohol (95%) q s ad | 1000 cc |
| Perfume and color              | q s     |
- (49) (Thomssen)
- |                       |         |
|-----------------------|---------|
| Sulfonated castor oil | 9.5 cc  |
| Oil of bergamot       | 1.1 cc  |
| Oil of cinnamon       | 0.1 cc  |
| Oil of clove          | 0.1 cc  |
| Oil of lavender       | 0.5 cc  |
| Tr of capsicum        | 0.8 cc  |
| Alcohol q s ad        | 1000 cc |

- (50) (Thomssen)  
 Sodium cetyl sulfate 20 Gm  
 Sulfonated castor oil 32 cc  
 Potassium arsenite 01 Gm  
 Alcohol 305 cc  
 Distilled water 622 cc  
 Perfume 02 cc

- (51) (Thomssen)  
 Fowler's solution 160 cc  
 Sulfonated castor oil 100 cc  
 Resorcinol monoacetate 15 cc  
 Perfume 025 cc  
 Alcohol q s ad 1000 cc

- (52) (Thomssen)  
 Glycerin 55 cc  
 Cholesterol 05 Gm  
 Lecithin 10 Gm  
 Distilled water 975 cc  
 Chloroform 80 cc  
 Perfume 025 cc  
 Alcohol q s ad 1000 cc

- (53) (Thomssen)  
 Cologne water essence 100 cc  
 Alcohol 700 cc  
 Glycerin 40 cc  
 Tr of cinchona 40 cc  
 Tr of quillaja 60 cc  
 Rose water 60 cc

## ALOPECIA AREATA

(Strongly Keratolytic and Rubefacient Preparations; Frequently Irritating and Sensitizing)

- (1) (Erasmus Wilson)  
 Stronger ammonia water 125 cc  
 Tr of cantharides 125 cc  
 Glycerin 125 cc  
 Distilled water q s ad 1000 cc

- (2) (Savill)  
 Tr of cantharides 125 cc  
 Acetic acid (dilute) 200 cc  
 Glycerin 125 cc  
 Alcohol 250 cc  
 Distilled water q s ad 1000 cc

- (3)  
 Tr of cantharides  
 Glycerin of each equal parts

- (4) (Stelwagon)  
 Oil of cade  
 Oil of turpentine  
 Olive oil of each equal parts

- (5) (Stelwagon)  
 Tr of cantharides  
 Tr of capsicum  
 Oil of turpentine of each equal parts  
 (Note: Watch this carefully dilute with almond or olive oil as indicated)

- (6)  
 Pure carbolic acid or 1 part of carbolic acid diluted in 3 parts of alcohol  
 (Note CAUTION!)

- (7) (Jackson)  
 Fluid extract of jaborandi boiled down to half its volume plus 4 parts of lard

- (8) (Hyde and Montgomery)  
 Carbolic acid 32 Gm  
 Tr of cantharides 125 cc  
 Castor oil 125 cc  
 Alcohol q s ad 1000 cc

- (9) (Jamison)  
 Resorcin 1020 Gm  
 White ointment q s ad 1000 Gm

- (10) (Sabouraud)  
 a) 25% glacial acetic acid in spirits of ether  
 b) Alcoholic solution of lactic acid 20%  
 c) 8% iodine in rectified benzine

- (11) (Lassar)  
 Wash head daily, then apply  
 a) Mercury bichloride 025 Gm  
 Glycerin 225 cc  
 Cologne water 225 cc  
 Distilled water q s ad 1000 cc  
 Then wash with  
 b) 05% alcoholic solution of naphthol and rub finally with  
 c) Salicylic acid 20 Gm  
 Tr of benzoin 30 cc  
 Neatsfoot oil q s ad 1000 cc

- (12) (Lassar)  
 Pilocarpine nitrate 20 Gm  
 Quinine hydrochloride 40 Gm  
 Precipitated sulfur 100 Gm  
 Balsam of Peru 200 Gm  
 Medulla bovina (Beef bone marrow) 1000 Gm

- (13) Tr of benzoin 120 cc  
 Spirits of ether q s ad 1000 cc

- (14) (J Neumann)  
 Carbolic acid liquefied 18 cc  
 Balsam of Peru 18 Gm  
 Spirit of lavender 18 cc  
 Spiritus vini gallici q s ad 1000 cc

- (15)  
 Glacial acetic acid 125 cc  
 Chloral hydrate 125 Gm  
 Ether q s ad 1000 cc  
 (Paint on bald spots)

- (16)  
 Tr of cantharides 64128 cc  
 Tr capsicum 128256 cc  
 Alcohol q s ad 1000 cc

- (17)
- |                   |         |
|-------------------|---------|
| Betanaphthol      | 30 Gm   |
| Salicylic acid    | 45 Gm   |
| Oil of lavender   | 06 cc   |
| Petrolatum q s ad | 1000 Gm |
- (Note Rub into bald spots at bedtime, then cover with cap [not rubber])

- (18)
- |                             |  |
|-----------------------------|--|
| Spirits of turpentine       |  |
| Oil of wintergreen          |  |
| Alcohol equal parts of each |  |
- (Note Paint on bald spots Beware of fire!)

- (19)
- |                           |          |
|---------------------------|----------|
| Resorcinol monoacetate    | 15 50 cc |
| Pilocarpine hydrochloride | 10 Gm    |
| Precipitated sulfur       | 100 Gm   |
| Balsam of Peru            | 40 Gm    |
| Lanolin                   | 40 Gm    |
| Distilled water           | 40 cc    |
| Petrolatum q s ad         | 1000 Gm  |

**ointments**

(Moderately Keratolytic and Rubefacient.  
Occasionally Irritating and Sensitizing)

- (1)
- |                           |         |
|---------------------------|---------|
| Pilocarpine hydrochloride | 25 Gm   |
| Oil of lavender           | 12 cc   |
| Lanolin                   | 750 Gm  |
| Petrolatum q s ad         | 1000 Gm |

- (2)
- |                         |           |
|-------------------------|-----------|
| Resorcin monoacetate    | 50 cc     |
| Balsam of Peru          | 50 Gm     |
| Precipitated sulfur     | 10 100 Gm |
| Benzoinated lard q s ad | 1000 Gm   |

- (3)
- |                     |          |
|---------------------|----------|
| Balsam of Peru      | 10 Gm    |
| Precipitated sulfur | 10 50 Gm |
| Salicylic acid      | 10 50 Gm |
| Lanolin             | 250 Gm   |
| Petrolatum q s ad   | 1000 Gm  |

- (4)
- |                   |          |
|-------------------|----------|
| Resorcin          | 10 30 Gm |
| Salicylic acid    | 10 30 Gm |
| Oil of rose       | 05 cc    |
| Petrolatum q s ad | 1000 Gm  |

- (5)
- |                     |           |
|---------------------|-----------|
| Salicylic acid      | 05 Gm     |
| Choral hydrate      | 10 50 Gm  |
| Precipitated sulfur | 50 100 Gm |
| Balsam of Peru      | 100 Gm    |
| Castor oil          | 100 cc    |
| Cocoa butter q s ad | 1000 Gm   |

- (6) (Goodman)
- |                           |         |
|---------------------------|---------|
| Pilocarpine hydrochloride | 10 Gm   |
| Distilled water           | 40 cc   |
| Precipitated sulfur       | 100 Gm  |
| Resorcinol monoacetate    | 40 cc   |
| Oxycholesterin base       | 40 Gm   |
| Balsam of Peru            | 40 Gm   |
| Lanolin                   | 160 Gm  |
| Petrolatum q s ad         | 1000 Gm |

- (7)
- |                        |         |
|------------------------|---------|
| Quinine hydrochloride  | 03 Gm   |
| Resorcinol monoacetate | 50 cc   |
| Lanolin                | 240 Gm  |
| Qualatum q s ad        | 1000 Gm |

- (8)
- |                             |         |
|-----------------------------|---------|
| Pilocarpine hydrochloride   | 20 Gm   |
| Balsam of Peru              | 20 Gm   |
| Castor oil                  | 20 cc   |
| Hydrophilic ointment q s ad | 1000 Gm |

**CHRONIC SCALP DISORDERS  
OF THE SCALP****Lotions**

- (1) (Savill)
- |                        |              |
|------------------------|--------------|
| Mercury bichloride     | 0 15 0 25 Gm |
| Coal tar solution      | 40 70 cc     |
| Sweet almond oil       | 0 4 60 cc    |
| Alcohol                | 40 200 cc    |
| Distilled water q s ad | 1000 cc      |

- (2) (Sabouraud)
- |                          |         |
|--------------------------|---------|
| Oil of cedar             | 80 cc   |
| Oil of cade (deodorized) | 80 cc   |
| Acetone                  | 280 cc  |
| Alcohol (95%) q s ad     | 1000 cc |

- (3) (Savill)
- |                        |           |
|------------------------|-----------|
| Salicylic acid         | 20 30 Gm  |
| Castor oil             | 0 4 30 cc |
| Spirits of rosemary    | 120 cc    |
| Alcohol                | 60 360 cc |
| Distilled water q s ad | 1000 cc   |

- (4) Dandruff Lotions
- |                        |         |
|------------------------|---------|
| Borax                  | 20 Gm   |
| Glycerin               | 100 cc  |
| Distilled water q s ad | 1000 cc |

- (5) (Thomssen)  
Dandruff Remover
- |                |        |
|----------------|--------|
| Betanaphthol   | 10 Gm  |
| Alcohol        | 489 cc |
| Tr of quillaja | 480 cc |
| Glycerin       | 20 cc  |
| Perfume        | 01 cc  |

- (6) (Savill)
- |                                |         |
|--------------------------------|---------|
| Mercury bichloride             | 03 Gm   |
| Coal tar solution              | 45 cc   |
| Phenol                         | 09 cc   |
| Castor oil                     | 10 cc   |
| Oil of lavender                | 03 cc   |
| Alcohol                        |         |
| Distilled water of each q s ad | 1000 cc |

- (7)
- |                      |         |
|----------------------|---------|
| Mercury bichloride   | 03 Gm   |
| Salicylic acid       | 20 Gm   |
| Chloral hydrate      | 20 Gm   |
| Oil of lavender      | 04 cc   |
| Oil of sweet almonds | 04 cc   |
| Acetone              | 250 cc  |
| Alcohol q s ad       | 1000 cc |

- (8)
- |                      |         |
|----------------------|---------|
| Chloral hydrate      | 10 Gm   |
| Tannic acid          | 10 Gm   |
| Sodium propionate    | 10 Gm   |
| Castor oil           | 50 cc   |
| Alcohol (90%) q s ad | 1000 cc |

- (9)
- |                |         |
|----------------|---------|
| Salicylic acid | 20 Gm   |
| Glycerin       | 10 cc   |
| Alcohol q s ad | 1000 cc |

- (10) (Sabouraud)
- |                         |         |
|-------------------------|---------|
| Carbon disulfide        | 1200 cc |
| Octahedral sulfur       | 100 Gm  |
| Carbon tetrachloride    | 1800 cc |
| Verberna                |         |
| Oil of bergamot of each | 10 cc   |

## Ointments

- (1) (Savill)
- |                                     |         |
|-------------------------------------|---------|
| Coal tar solution                   | 60 cc   |
| Ammoniated mercury                  | 10 Gm   |
| White petrolatum q s ad             | 1000 Gm |
| (Salicylic acid 10.30 can be added) |         |

- (2) (Savill)
- |                   |         |
|-------------------|---------|
| Coal tar solution | 250 cc  |
| Lard q s ad       | 1000 Gm |

- (3) (Sabouraud)
- a) Oil of cade (deodorized)
- |                                            |        |
|--------------------------------------------|--------|
| Lanolin                                    | 100 cc |
| White petrolatum                           | 100 Gm |
| Pyrogallic acid                            | 10 Gm  |
| Turpethum minerale (Basic mercury sulfate) | 10 Gm  |
- and
- b) Neutral washed coal tar
- |                    |        |
|--------------------|--------|
| Lanolin            | 60 cc  |
| White petrolatum   | 60 Gm  |
| Ammoniated mercury | 200 Gm |
| Resorcin           | 10 Gm  |

- and
- c) Clear off in the morning with
- |                                     |  |
|-------------------------------------|--|
| Alcohol (95%)                       |  |
| Acetone anhydrous                   |  |
| Distilled water of each equal parts |  |

- (4)
- |                           |          |
|---------------------------|----------|
| Ammoniated mercury        | 10.50 Gm |
| Salicylic acid            | 10.50 Gm |
| Cold cream                |          |
| Petrolatum of each q s ad | 1000 Gm  |

- (5) (Bulkley)
- |                             |            |
|-----------------------------|------------|
| Ammoniated mercury ointment | 250.500 Gm |
| Rose water ointment         | 600 Gm     |

- (6)
- |                             |          |
|-----------------------------|----------|
| Precipitated sulfur         | 20.50 Gm |
| Salicylic acid              | 20.50 Gm |
| Hydrophilic ointment q s ad | 1000 Gm  |

- (7)
- |                        |          |
|------------------------|----------|
| Deodorized oil of cade | 10.50 cc |
| Salicylic acid         | 10.50 Gm |
| Lanolin                | 250 Gm   |
| Petrolatum q s ad      | 1000 Gm  |

## SEBORRHEA OLEOSA AND SEBORRHEIC DERMATITIS

## Ointments

- (1)
- |                   |         |
|-------------------|---------|
| Sublimated sulfur | 30 Gm   |
| Salicylic acid    | 20 Gm   |
| Balsam of Peru    | 20 Gm   |
| Petrolatum q s ad | 1000 Gm |

- (2) (Bulkley)
- |                            |           |
|----------------------------|-----------|
| Resorcin                   | 40.120 Gm |
| Zinc oxide                 | 6.5 Gm    |
| Rose water ointment q s ad | 1000 Gm   |

- (3)
- |                         |         |
|-------------------------|---------|
| Ammoniated mercury      | 120 Gm  |
| Benzoinated lard q s ad | 1000 Gm |

- (4)
- |                             |         |
|-----------------------------|---------|
| Ammoniated mercury          | 120 Gm  |
| Yellow oxide of mercury     | 30 Gm   |
| Salicylic acid              | 30 Gm   |
| Hydrophilic ointment q s ad | 1000 Gm |

- (5)
- |                         |         |
|-------------------------|---------|
| Ammoniated mercury      | 120 Gm  |
| Oil of cade             | 120 cc  |
| Benzoinated lard q s ad | 1000 Gm |

- (6)
- |                         |         |
|-------------------------|---------|
| Ammoniated mercury      | 120 Gm  |
| Yellow oxide of mercury | 30 Gm   |
| Salicylic acid          | 30 Gm   |
| Oleoresin of capsicum   | 0.3 cc  |
| Oil of cade             | 120 cc  |
| Hydrophilic ointment ad | 1000 Gm |

- (7) (Goodman)
- |                     |         |
|---------------------|---------|
| Sulfur              | 100 Gm  |
| Glycerin            | 100 cc  |
| Salicylic acid      | 20 Gm   |
| Lard                | 100 Gm  |
| Cocoa butter q s ad | 1000 Gm |

- (8)
- |                            |         |
|----------------------------|---------|
| Mercury olea e             | 7.5 Gm  |
| Menthol                    | 0.5 Gm  |
| Pine needle oil            | 10 cc   |
| Light liquid petrolatum ad | 1000 cc |

- (9)
- |                             |         |
|-----------------------------|---------|
| For Chronic Resistant Types |         |
| Coal tar solution           | 2.5 cc  |
| Precipitated sulfur         |         |
| Salicylic acid of each      | 2.5 Gm  |
| Hydrophilic petrolatum ad   | 1000 Gm |



|      |                        |         |
|------|------------------------|---------|
| (10) |                        |         |
|      | Oil of cade            | 25 cc   |
|      | Ammoniated mercury     |         |
|      | Salicylic acid of each | 25 Gm   |
|      | Hydrophilic ointment   | 500 Gm  |
|      | Petrolatum q s ad      | 1000 Gm |

|      |                        |          |
|------|------------------------|----------|
| (11) |                        |          |
|      | Resorcin monoacetate   | 10.50 cc |
|      | Precipitated sulfur    |          |
|      | Salicylic acid of each | 10.50 Gm |
|      | Castor oil             | 100 cc   |
|      | Aquaphor q s ad        | 1000 Gm  |

|      |                        |          |
|------|------------------------|----------|
| (12) |                        |          |
|      | Sodium undecylenate    |          |
|      | or                     |          |
|      | Sodium propionate      | 10.50 Gm |
|      | Precipitated sulfur    | 10.30 Gm |
|      | Oil of rose            | 05 cc.   |
|      | Hydrophilic petrolatum |          |
|      | ad                     | 1000 Gm  |

|      |                        |          |
|------|------------------------|----------|
| (13) |                        |          |
|      | For Resistant Cases    |          |
|      | Vioform                | 10.30 Gm |
|      | Coal tar solution      | 05.30 cc |
|      | Hydrophilic petrolatum |          |
|      | q s ad                 | 1000 Gm  |

|      |                         |         |
|------|-------------------------|---------|
| (14) |                         |         |
|      | Diiodo hydroxyquinoline |         |
|      | (Diodoquin)             | 20 Gm   |
|      | Coal tar solution       | 20 cc   |
|      | Hydrophilic ointment    |         |
|      | q s ad                  | 1000 Gm |

|      |                     |          |
|------|---------------------|----------|
| (15) |                     |          |
|      | For Resistant Cases |          |
|      | Salicylic acid      | 10.50 Gm |
|      | Pyrogallol          | 10.50 Gm |
|      | Castor oil          | 100 cc   |
|      | Petrolatum q s ad   | 1000 Gm  |

|      |                       |          |
|------|-----------------------|----------|
| (16) |                       |          |
|      | Cinnabar              | 05.20 Gm |
|      | Precipitated sulfur   | 10.30 Gm |
|      | Castor oil            | 100 cc   |
|      | White ointment q s ad | 1000 Gm  |

|      |                                                                                                                                                  |          |
|------|--------------------------------------------------------------------------------------------------------------------------------------------------|----------|
| (17) |                                                                                                                                                  |          |
|      | Chrysarobin                                                                                                                                      | 10.50 Gm |
|      | Salicylic acid                                                                                                                                   | 10.30 Gm |
|      | Petrolatum q s ad                                                                                                                                | 1000 Gm  |
|      | (Note: Chrysarobin can be used in the scalp but only with the greatest caution and for chronic inveterate cases resistant to all other therapy.) |          |

|      |                       |          |
|------|-----------------------|----------|
| (18) |                       |          |
|      | Oil of cade           | 20.80 cc |
|      | Precipitated sulfur   | 20.50 Gm |
|      | Salicylic acid        | 10.30 Gm |
|      | Castor oil            | 50 cc    |
|      | White ointment q s ad | 1000 Gm  |

(For additional prescriptions see *Psoriasis of the Scalp* page 446.)

## Lotions

|     |                    |        |
|-----|--------------------|--------|
| (1) | (Savill)           |        |
|     | Tannic acid        | 15 Gm  |
|     | Spirit of lavender | 500 cc |
|     | Spirit of rosemary | 500 cc |

|     |                   |           |
|-----|-------------------|-----------|
| (2) | (Bulkley)         |           |
|     | Resorcin          | 32.96 Gm  |
|     | Alcohol           | 64.120 cc |
|     | Glycerin          | 70.128 cc |
|     | Rose water q s ad | 1000 cc   |

|     |                        |         |
|-----|------------------------|---------|
| (3) |                        |         |
|     | Resorcinol monoacetate | 24 cc   |
|     | Mercury bichloride     | 004 Gm  |
|     | Salicylic acid         | 16 Gm   |
|     | Glycerin               | 20 cc   |
|     | Spirit of lavender     | 40 cc   |
|     | Alcohol (70%) q s ad   | 1000 cc |

|     |                        |         |
|-----|------------------------|---------|
| (4) |                        |         |
|     | Sodium propionate      | 12 Gm   |
|     | Resorcinol monoacetate | 20 cc   |
|     | Salicylic acid         | 16 Gm   |
|     | Castor oil             | 20 cc   |
|     | Cologne water          | 500 cc  |
|     | Alcohol (70%) q s ad   | 1000 cc |

## BACTERICIDAL AND ANTISEPTIC PREPARATIONS

|     |                           |         |
|-----|---------------------------|---------|
| (1) |                           |         |
|     | Oxyquinoline sulfate      | 05 Gm   |
|     | Benzoyl peroxide          | 100 Gm  |
|     | Thymol                    | 065 Gm  |
|     | Eucalyptol                | 13 cc   |
|     | Petrolatum                |         |
|     | Anhydrous lanolin of each |         |
|     | ad                        | 1000 Gm |

|     |                         |         |
|-----|-------------------------|---------|
| (2) |                         |         |
|     | Comp. quinolor ointment | 200 Gm  |
|     | Precipitated sulfur     | 20 Gm   |
|     | Castor oil              | 50 cc   |
|     | Petrolatum q s ad       | 1000 Gm |

|     |                     |         |
|-----|---------------------|---------|
| (3) |                     |         |
|     | Ammoniated mercury  | 50 Gm   |
|     | Salicylic acid      | 10 Gm   |
|     | Rose water ointment |         |
|     | q s ad              | 1000 Gm |

|     |                     |         |
|-----|---------------------|---------|
| (4) |                     |         |
|     | Vioform             | 20 Gm   |
|     | Bacitracin ointment |         |
|     | or                  |         |
|     | Petrolatum q s ad   | 1000 Gm |

|     |                         |         |
|-----|-------------------------|---------|
| (5) |                         |         |
|     | Diiodo hydroxyquinoline |         |
|     | (Diodoquin)             | 20 Gm   |
|     | Bacitracin ointment     |         |
|     | or                      |         |
|     | Petrolatum q s ad       | 1000 Gm |

|     |                     |  |
|-----|---------------------|--|
| (6) |                     |  |
|     | Bacitracin ointment |  |

- (7) Aureomycin or terramycin ointment
- (8) Comp quinolor ointment 18 0 Gm  
Bacitracin ointment q s ad 100 0 Gm
- (9) Hexachlorophene 1 0 Gm.  
Mercury bichloride 0 06 Gm  
Salicylic acid 3 0 Gm  
Resorcinol monoacetate 3 0 cc  
Spirit of lavender 15 0 cc  
Castor oil 5 0 cc  
Alcohol (70%) q s ad 100 0 cc

## FUNGICIDES

- (1) Sodium undecylenate  
Sodium caprylate  
Sodium propionate of each 3 0 10 0 Gm  
Hydrophilic ointment q s ad 100 0 Gm
- (2) Salicylic acid 2 0 Gm.  
Ammoniated mercury 2 0 Gm  
Hydrophilic ointment q s ad 100 0 Gm
- (3) Precipitated sulfur 3 0 Gm  
Salicylic acid 3 0 Gm.  
Coal tar solution 12 0 cc  
Lanolin 15 0 Gm.  
Hydrophilic petrolatum q s ad 100 0 Gm
- (4) Salicylanilid 5 0 Gm  
Hyamine 1622, 25% 5 0 Gm  
Hydrophilic ointment q s ad 100 0 Gm
- (5) Salicylic acid 4 5 Gm  
Precipitated sulfur 9 0 Gm  
Oil of cade 15 0 cc  
Hydrophilic petrolatum q s ad 100 0 Gm
- (6) Salicylic acid 1 0 5 0 Gm  
Benzoic acid 2 0 10 0 Gm  
Petrolatum q s ad 100 0 Gm
- (7) Precipitated sulfur 3 0 Gm  
Salicylic acid 3 0 Gm  
Water washable ointment base q s ad 100 0 Gm
- (8) Iodine crystals 0 5 5 0 Gm  
Thymol 0 5 5 0 Gm  
Lanolin 45 0 Gm  
Petrolatum q s ad 100 0 Gm

- (9) Salicylic acid 3 0 Gm  
Mineral oil 6 0 cc  
Tween 60 1 0 Gm  
Span 60 1 0 Gm  
Lanolin q s ad 100 0 Gm

- (10) Salol 1 5 Gm  
Thymol 1 8 Gm  
Salicylic acid 4 0 Gm  
Benzoic acid 4 0 Gm  
Mineral oil 12 0 cc  
Lanolin 20 0 Gm  
Petrolatum q s ad 100 0 Gm

- (11) (Reuss and Doherty)  
Podophyllin 0 2 Gm  
Carbowax q s ad 100 0 Gm

(For additional prescriptions see section on fungus infections of the scalp, page 323 )

## PRURITUS

### Ointments and Lotions

- (1) Menthol 0 2 Gm  
Phenol 1 0 Gm  
Hydrophilic ointment ad 100 0 Gm
- (2) Aluminum subacetate solution 15 0 cc  
Anhydrous lanolin 30 0 Gm  
Hydrophilic ointment q s ad 100 0 Gm
- (3) Eurax ointment q s
- (4) Antihistamine powder 1 0 5 0 Gm  
Water washable ointment q s ad 100 0 Gm  
(Note Many such preparations are commercially available)
- (5) Menthol 0 2 Gm  
Benzocaine 3 0 Gm  
Cold cream, unperfumed q s ad 100 0 Gm
- (6) Menthol 0 2 Gm  
Coal tar solution 5 0 cc  
Distilled water q s ad 100 0 cc
- (7) Benzocaine 1 0 Gm  
Chloral hydrate 4 0 Gm  
Castor oil 5 0 cc  
Alcohol 20 0 cc  
Distilled water q s ad 100 0 cc.
- (8) Phenol 1 0 Gm  
Camphor 3 0 Gm  
Glycerin q s ad 100 0 cc

- |               |                                    |            |                                                |                                           |             |
|---------------|------------------------------------|------------|------------------------------------------------|-------------------------------------------|-------------|
| (9)           | Menthol                            | 0.2 Gm     | (4) Anthralin                                  | 0.1 1.0 Gm                                |             |
|               | Coal tar solution                  | 60 cc      | Water washable base q s ad                     | 100.0 Gm                                  |             |
|               | Lotion Solucreme q s ad            | 100.0 cc   | (Note Anthralin discolors skin and light hair) |                                           |             |
| (10)          | Menthol                            | 0.1 Gm     | (5)                                            | Ammoniated mercury                        | 5.0 10.0 Gm |
|               | Boric acid                         | 3.0 Gm     |                                                | Salicylic acid                            | 5.0 Gm      |
|               | Distilled water q s ad             | 100.0 cc   |                                                | Lanolin                                   | 30.0 Gm     |
|               |                                    |            |                                                | Petrolatum q s ad                         | 100.0 Gm    |
| (11) (Savill) |                                    |            | (6)                                            | Coal tar solution                         | 5.0 50.0 cc |
|               | Menthol                            | 1.0 Gm     |                                                | Acetone                                   |             |
|               | Alcohol                            | 25.0 cc    |                                                | Alcohol of each ad                        | 100.0 cc    |
|               | Acetic acid dilute                 | 6.0 cc     |                                                | (Note Paint on patches morning and night) |             |
|               | Distilled water q s ad             | 100.0 cc   | (7)                                            |                                           |             |
| (12) (Savill) |                                    |            |                                                | Mercury bichloride                        | 0.1 Gm      |
|               | Dilute solution of lead subacetate | 12.0 cc    |                                                | Resorcinol monoacetate                    | 3.0 cc      |
|               | Coal tar solution                  | 6.0 cc     |                                                | Salicylic acid                            | 1.0 Gm      |
|               | Distilled water q s ad             | 100.0 cc   |                                                | Spirit of lavender                        | 15.0 cc     |
| (13) (Savill) |                                    |            |                                                | Castor oil                                | 5.0 cc      |
|               | Coal tar solution                  | 1 in 10    |                                                | Cologne water                             | 30.0 cc     |
|               |                                    |            |                                                | Alcohol (50%) q s ad                      | 100.0 cc    |
| (14)          |                                    |            | (8)                                            |                                           |             |
|               | Carbolic acid                      | 0.5 2.0 cc |                                                | Pyrogallol                                | 5.0 10.0 Gm |
|               | Distilled water q s ad             | 100.0 cc   |                                                | Ether or acetone                          | 50.0 cc     |
|               |                                    |            |                                                | Alcohol q s ad                            | 100.0 cc    |

**PSORIASIS OF THE SCALP**

- |                                                                    |                                             |             |      |                  |          |
|--------------------------------------------------------------------|---------------------------------------------|-------------|------|------------------|----------|
| (1) (Jadassohn)                                                    |                                             |             | (9)  | Salicylic acid   | 10.0 Gm  |
|                                                                    | Coal tar solution                           | 2.0 20.0 cc |      | Pyrogallol       | 10.0 Gm  |
|                                                                    | Ammoniated mercury                          | 5.0 10.0 Gm |      | Liquid pine tar  | 15.0 cc  |
|                                                                    | Lanolin                                     | 50.0 Gm     |      | Lanolin          | 25.0 Gm  |
|                                                                    | Olive oil                                   | 10.0 cc     |      | Soft soap q s ad | 100.0 Gm |
|                                                                    | Distilled water q s ad                      | 100.0 cc    | (10) |                  |          |
| (2) (Sabouraud's favorite prescription for psoriasis of the scalp) |                                             |             |      | Pyrogallol       | 6.0 Gm   |
| a                                                                  | Oil of cade (deodorized)                    | 10.0 cc     |      | Salicylic acid   | 4.0 Gm   |
|                                                                    | Lanolin                                     | 10.0 Gm     |      | Liquid pine tar  | 20.0 cc  |
|                                                                    | White petrolatum                            | 10.0 Gm     |      | Ichthammol       | 20.0 Gm  |
|                                                                    | Luprithum minerale                          |             |      | White wax q s ad | 100.0 Gm |
|                                                                    | Mercurii subsulfate (yellow)                | 1.0 Gm      |      |                  |          |
|                                                                    | Pyrosulfuric acid                           | 1.0 Gm      |      |                  |          |
|                                                                    | and                                         |             |      |                  |          |
| b                                                                  | Neutral washed coal tar                     | 6.0 cc      |      |                  |          |
|                                                                    | Lanolin                                     | 6.0 Gm      |      |                  |          |
|                                                                    | White petrolatum                            | 20.0 Gm     |      |                  |          |
|                                                                    | Ammoniated mercury                          | 1.0 Gm      |      |                  |          |
|                                                                    | Resorcin                                    | 1.0 Gm      |      |                  |          |
|                                                                    | and                                         |             |      |                  |          |
| c                                                                  | Clear off in the morning with Alcohol (95%) |             |      |                  |          |
|                                                                    | Acetone anhydrous                           |             |      |                  |          |
|                                                                    | Distilled water of each equal parts         |             |      |                  |          |
| (3)                                                                |                                             |             |      |                  |          |
|                                                                    | Pyrogallol                                  | 5.0 Gm      |      |                  |          |
|                                                                    | Salicylic acid                              | 3.0 Gm      |      |                  |          |
|                                                                    | Liquid pine tar                             | 10.0 cc     |      |                  |          |
|                                                                    | Lanolin                                     | 25.0 Gm     |      |                  |          |
|                                                                    | Petrolatum q s ad                           | 100.0 Gm    |      |                  |          |

(Also see prescriptions 13 18 under *Seborrhea Oleosa* and *Seborrheic Dermatitis* page 550.)

**HAIR DRESSINGS**

- |                  |                   |          |
|------------------|-------------------|----------|
| (1) (Max Joseph) |                   |          |
|                  | Lard              | 60.0 Gm  |
|                  | White wax         | 12.0 Gm  |
|                  | Oil of violet     | 4.0 cc   |
|                  | Oil of heliotrope | 15.0 cc  |
| (2)              |                   |          |
|                  | Cocoa butter      | 25.0 Gm  |
|                  | Gumme sulfate     | 3.0 Gm   |
|                  | Oil of rose       | 0.8 cc   |
|                  | Almond oil q s ad | 100.0 cc |
| (3)              |                   |          |
|                  | Tr of benzoin     | 3.0 cc   |
|                  | Balsam of Peru    | 6.0 Gm   |
|                  | Almond oil        | 12.0 cc  |
|                  | Lard q s ad       | 100.0 Gm |

- |                           |         |  |                          |         |  |
|---------------------------|---------|--|--------------------------|---------|--|
| (4) (Goodman)             |         |  | (5) (Harry)              |         |  |
| Resorcin monoacetate      | 20 cc   |  | Liquid petrolatum        | 230 cc  |  |
| Salicylic acid            | 20 Gm   |  | Triethanolamine stearate | 50 cc   |  |
| Precipitated sulfur       | 40 Gm   |  | Distilled water q s ad   | 1000 cc |  |
| Ceresin                   | 240 Gm  |  | Perfume and preservative | q s     |  |
| Coconut oil q s ad        | 1000 cc |  |                          |         |  |
| (5) (Goodman)             |         |  | (6) (Harry)              |         |  |
| Pilocarpine hydrochloride | 20 Gm   |  | Deodorized kerosene      | 100 cc  |  |
| Distilled water           | 20 cc   |  | Liquid petrolatum        | 200 cc  |  |
| Oil of rose               | 02 cc   |  | Diglycol laurate         | 140 Gm  |  |
| Sweet almond oil          | 500 cc  |  | Distilled water q s ad   | 1000 cc |  |
| Cocoa butter q s ad       | 1000 Gm |  | Perfume and preservative | q s     |  |
| (6) (Goodman)             |         |  | (7) (Harry)              |         |  |
| Resorcinol monoacetate    | 40 cc   |  | Glyceryl monostearate    | 80 Gm   |  |
| Castor oil                | 300 cc  |  | Sapamine citrate         | 08 Gm   |  |
| Sesame oil                | 300 cc  |  | Lanolin                  | 25 Gm   |  |
| Spermaceti q s ad         | 1000 Gm |  | Cholesterol              | 05 Gm   |  |
| (7) (Sabouraud)           |         |  | Distilled water q s ad   | 1000 cc |  |
| Oil of cade               | 500 cc  |  | Lemon juice              |         |  |
| Oil of birch              | 50 cc   |  | Perfume                  |         |  |
| Ichthammol                | 50 Gm   |  | Preservative of each     | q s     |  |
| Resorcin                  | 50 Gm   |  |                          |         |  |
| Lanolin                   | 1000 Gm |  | (8) (Harry)              |         |  |
| (8)                       |         |  | Setting Lotion           |         |  |
| Liquid petrolatum         | 100 cc  |  | Gum tragacanth           | 12 Gm   |  |
| Petrolatum q s ad         | 1000 Gm |  | Alcohol                  | 150 cc  |  |
| Perfume                   | q s     |  | Glycerin                 | 20 cc   |  |
|                           |         |  | Distilled water q s ad   | 1000 cc |  |
|                           |         |  | Preservative             | q s     |  |
|                           |         |  | (9) (Harry)              |         |  |
|                           |         |  | Hair Lacquer             |         |  |
|                           |         |  | Rosin                    | 10 Gm   |  |
|                           |         |  | Gum benzoin              | 20 Gm   |  |
|                           |         |  | Alcohol q s ad           | 1000 cc |  |
|                           |         |  | Perfume                  | q s     |  |
|                           |         |  | (10) (de Navarre)        |         |  |
|                           |         |  | Hair Lacquer             |         |  |
|                           |         |  | Tr of benzoin            | 200 cc  |  |
|                           |         |  | Alcohol S D q s ad       | 1000 cc |  |
|                           |         |  | Color and perfume        | q s     |  |

# **NONOILY FIXATIVES AND HAIR CREAMS**

- |                                     |         |  |
|-------------------------------------|---------|--|
| (1) (Harry)                         |         |  |
| Gum tragacanth (powdered)           | 10 Gm   |  |
| Alcohol                             | 60 cc   |  |
| Castor oil                          | 20 cc   |  |
| Distilled water                     | 900 cc  |  |
| Glycerin                            | 10 cc   |  |
| Preservative                        | q s     |  |
| (2) (Harry)                         |         |  |
| Gum karaya                          | 20 Gm   |  |
| Alcohol                             | 50 cc   |  |
| Distilled water                     | 930 cc  |  |
| Perfume and preservative            | q s     |  |
| (3) (Harry)                         |         |  |
| Sodium alginate                     | 12 Gm   |  |
| Calcium citrate                     | 02 Gm   |  |
| Alcohol                             | 30 cc   |  |
| Perfume                             | 05 cc   |  |
| Distilled water                     | 952 cc  |  |
| Preservative                        | q s     |  |
| (4) (Harry)                         |         |  |
| Petrolatum                          | 75 Gm   |  |
| Mineral oil 65/75                   | 375 cc  |  |
| Anhydrous lanolin                   | 30 Gm   |  |
| Arlacel C (Sorbitan<br>sesquiolate) | 30 Gm   |  |
| Borax                               | 20 Gm   |  |
| Borax                               | 05 Gm   |  |
| Distilled water q s ad              | 1000 cc |  |

# **HAIR OILS**

- |                                    |         |  |
|------------------------------------|---------|--|
| (1)                                |         |  |
| Sweet almond oil                   | 660 cc  |  |
| Oil of jasmine                     | 340 cc  |  |
| Oil of rose                        | 006 cc  |  |
| (2)                                |         |  |
| Oil of jasmine                     |         |  |
| Oil of rose of each                | 300 cc  |  |
| Oil of orange flowers              |         |  |
| Oil of acacia                      |         |  |
| Oil of Polyanth tuberos<br>of each | 150 cc  |  |
| Oil of hyacinth                    |         |  |
| Oil of narcissus                   |         |  |
| Aromatic vanilla Tr<br>of each     | 75 cc   |  |
| (3)                                |         |  |
| Oil of rose geranium               | 20 cc   |  |
| Oil of rosemary                    | 50 cc   |  |
| Oil of lavender                    | 120 cc  |  |
| Oil of lemon                       | 200 cc  |  |
| Sesame oil q s ad                  | 1000 cc |  |

- (4)
- |                      |         |
|----------------------|---------|
| Oil of rose geranium | 50 cc   |
| Oil of bergamot      | 100 cc  |
| Oil of lavender      | 150 cc  |
| Sunflower oil q s ad | 1000 cc |

**BRILLIANTINES****Liquid**

- (1)
- |                 |         |
|-----------------|---------|
| Olive oil       | 350 cc  |
| Mineral oil     | 200 cc  |
| Oil of lavender | 10 cc   |
| Alcohol q s ad  | 1000 cc |
- (2)
- |                |         |
|----------------|---------|
| Mineral oil    | 350 cc  |
| Sesame oil     | 200 cc  |
| Alcohol q s ad | 1000 cc |
- (3)
- |                   |         |
|-------------------|---------|
| Mineral oil       | 750 cc  |
| Sweet almond oil  | 100 cc  |
| Castor oil q s ad | 1000 cc |
| Perfume           | q s     |
- (4)
- |                |         |
|----------------|---------|
| Resorcin       | 0.06 Gm |
| Castor oil     | 300 cc  |
| Oil of violet  | 10 cc   |
| Alcohol q s ad | 1000 cc |
- (5) (Goodman)
- |                |         |
|----------------|---------|
| Castor oil     | 40 cc   |
| Almond oil     | 240 cc  |
| Glycerin       | 100 cc  |
| Alcohol q s ad | 1000 cc |
- (6) (de Navarre)
- |                          |         |
|--------------------------|---------|
| Methyl linoleate         | 10 Gm   |
| Ethyl myristate          | 250 Gm  |
| Mineral oil 85/95 q s ad | 1000 cc |
| Color and perfume        | q s     |
- (7) (de Navarre)
- |                          |         |
|--------------------------|---------|
| Refined oleyl alcohol    | 150 cc  |
| Mineral oil 85/95 q s ad | 1000 cc |
| Perfume                  | q s     |
- (8) (de Navarre)
- |                          |         |
|--------------------------|---------|
| Propylene glycol laurate | 100 Gm  |
| Mineral oil 85/95 q s ad | 1000 cc |
| Perfume                  | q s     |
- (9)
- |                               |         |
|-------------------------------|---------|
| Parahydroxybenzoic acid ester | 0.05 Gm |
| Oil of bergamot               | 10 cc   |
| Castor oil                    | 50 cc   |
| Olive oil                     | 450 cc  |
| Sweet almond oil q s ad       | 1000 cc |
- (10)
- |                           |         |
|---------------------------|---------|
| Odorless kerosene         | 200 cc  |
| Olive oil                 | 300 cc  |
| Oil of lavender           | 10 cc   |
| Mineral oil, light q s ad | 1000 cc |

- (11)
- |                             |         |
|-----------------------------|---------|
| <b>Nonoily Brilliantine</b> |         |
| Glycerin                    | 450 cc  |
| Alcohol (50%) q s ad        | 1000 cc |
| Perfume                     | q s     |

- (12) (Harry)
- |                        |         |
|------------------------|---------|
| Olive oil (deodorized) | 100 cc  |
| Avocado oil            | 250 cc  |
| Liquid paraffin q s ad | 1000 cc |
| Perfume and color      | q s     |
- (13) (Harry)
- |                        |         |
|------------------------|---------|
| Apricot kernel oil     | 650 cc  |
| Olive oil (deodorized) | 150 cc  |
| Castor oil             | 20 cc   |
| Alcohol q s ad         | 1000 cc |
| Perfume                | q s     |

**Solid and Semisolid**

- (1)
- |                         |         |
|-------------------------|---------|
| Castor oil              | 30 cc   |
| Goose grease            | 30 Gm   |
| Cocoa butter            | 120 Gm  |
| White wax               | 60 Gm   |
| Lanolin                 | 250 Gm  |
| Oil of rose geranium    | 08 cc   |
| White petrolatum q s ad | 1000 Gm |
- (2)
- |                    |         |
|--------------------|---------|
| Petrolatum         | 500 Gm  |
| Ozokerite          | 80 Gm   |
| Oil of bergamot    | 10 cc   |
| Mineral oil q s ad | 1000 cc |
- (3)
- |                   |         |
|-------------------|---------|
| Oil of neroli     | 10 cc   |
| Olive oil         | 650 cc  |
| Spermaceti q s ad | 1000 Gm |
- (4)
- |                    |         |
|--------------------|---------|
| Spermaceti         | 40 Gm   |
| Stearic acid       | 100 Gm  |
| Paraffin           | 150 Gm  |
| Oil of lavender    | 10 cc   |
| Mineral oil q s ad | 1000 cc |
- (5) (Thomssen)
- |                          |         |
|--------------------------|---------|
| Spermaceti               | 150 Gm  |
| Myristic acid            | 50 cc   |
| Oleic acid               | 240 cc  |
| White mineral oil q s ad | 1000 cc |
| Perfume                  | q s     |
- (6) (Thomssen)
- |                          |         |
|--------------------------|---------|
| Stearic acid             | 90 Gm   |
| Spermaceti               | 100 Gm  |
| Sweet almond oil         | 300 cc  |
| Perfume                  | 10 cc   |
| White mineral oil q s ad | 1000 cc |
- (7)
- |                    |         |
|--------------------|---------|
| Ceresin            | 50 Gm   |
| Petrolatum         | 300 Gm  |
| Coconut oil q s ad | 1000 cc |
| Perfume            | q s     |

# INDEX

## A

- Aberrant lesions of scalp with other diseases 450
- Abscesses with cellulitis of scalp 158
- Absorption percutaneous and permeability of skin and scalp 92
- Acarophobia 392
- Achorion (Trichophyton) schoenleii 332 336 337
- Acne keloid (*see* Folliculitis keloidalis)
  - necrotic miliaris 407
  - urticata 408
  - various forms 409
  - therapy 411
  - vulgaris lesions of scalp with 450
- Actins 268 269
- Acrodermatitis continua 412
- ACTH and hair growth 105
- n treatment of dermatitis of scalp 159
  - of lupus erythematosus 466
  - dysmaturus 467
  - of psoriasis 449
  - of scleroderma 472

181

- Albinism 74 75
- Alopecia 44 72
  - and pyoderma secondary to pediculosis capitis 347
  - androgens and 229
  - anomalies associated with 168
  - areata clinical features 357
    - different diagnosis 363
    - differentiated from tinea capitis 290
  - etiology 368
  - histology 367
  - ophiasis type 366
  - pathology 365
  - preparations formulary 547
  - theories as to cause 368 373
  - therapy 373
  - typical hairs 360
  - with neurodermatitis 389
- caused by arsenic 420
  - by atabrine 420
  - by atomic bombs 19
  - by barbiturates 421
  - by bromides 421
  - by cyanoacetic hydrochloride 421 427
  - by gold therapy 421
  - by iodides 421
  - by propylthiouracil 421

- Alopecia caused—Cont'd
  - by psoriasis 441
  - by quinacrine hydrochloride 421
  - by roentgen and radium rays 189
  - by thallium salts 421
  - causes of 229
  - catagenic 170 171
    - and follicular plugging of scalp 433
    - different features of 214
    - due to physical agents 181
    - due to seborrhea dermatitis 230
    - following favus 333
    - following kerion 321
    - following x-ray therapy 190 191
    - with folliculitis decalvans 209 210 211
  - classification 162
    - of scalps 174
  - congenital classification 164
    - clinical features 163
    - etiology 167
    - pathology 166
    - syndrome 165
    - therapy 167
    - triangular 166
  - diffuse with extensive neurodermatitis 379
  - due to congenital syphilis 280 281 282
  - female pattern 178
  - chthiosis with 427
  - in association with systemic diseases 457
  - in mycosis fungoides 534 535
  - in Vogt-Koyanagi syndrome 473
  - linear frontal 185
  - male pattern 173
    - aging as factor 181
    - clinical features 173
    - endocrines and etiology of 181
    - etiology 178
    - hereditary 181
    - in women 177
    - pathology 178
    - therapy 183
    - with seborrhea oleosa 227
  - moth eaten of syphilis 273 274 275
  - noncatagenic 412
  - nonspecific 412
  - ophiasis type with sickle cell anemia 473
  - partial due to trichotillomania 386
  - permanent resulting from perifolliculitis and pyoderma 250
  - pseudopelade 200
  - temporary following superficial folliculitis 250
  - total 364 368
    - due to congenital syphilis 279
  - traumatic marginal 185 186 187
  - triangular congenital 166

- Alopecia**—Cont d  
 with keratosis blennorrhagica 428  
 with lupus erythematosus 460  
 disseminatus 467
- Amol** 146
- Amne or anline type of synthetic organ**  
 hair dyes 146
- Amnion** in treatment of psoriasis 450
- Anatomy of hair follicle** 34  
 shaft 35  
 of scalp 31  
 of sebaceous glands 49  
 of sweat glands 55
- Androgens and alopecia** 229  
 and hair growth 99
- Anemopernicous** 467
- Scale cell** 472 473
- Anesthesia** in treatment of neurodermatitis 386  
 local for surgery of scalp 478
- Angioma (vascular nevi)** 497  
 cavernous 498  
 simplex (strawberry mark) 497 498
- Anhidrotic ectodermal dysplasia** hereditary 186
- Anline type of synthetic organic hair dyes** 146
- Animal hairs** medical aspects 194 195  
 parasites 316
- Anthrax** 265

6

- of seborrheic dermatitis 243
- Antistaminics** in treatment of pruritus 402
- Antiseptic and bactericidal preparations**  
 formulary 550
- Apocrine sweat glands** anatomy of 56
- Areas of cross section of hair** 70
- Arrangement of hair on body** 61
- Arsenic** as cause of dermatitis 420
- Arsenical keratosis** 483
- Arterial supply of scalp** 57
- Artificial waving** 137
- Atabine** as cause of dermatitis 420
- Atomic bomb** effect of on scalp 19
- Atopodermia** 414
- Atrophic** dead hairs 360
- Auricular fistula** congenital 171 17
- Axils** on scalp hair by electric drill 185
- Axilation of scalp** surgical considerations 477
- Azotoprene** of synthetic organic hair dyes 145

## B

- Bacillus anthracis** 265
- Bacterial infections of scalp** 45  
 acne keloid 56  
 anthrax 65  
 carbuncle 49  
 dermatitis papillaris agilis 51  
 disseminating pustules 6  
 erysipelas 66  
 folliculitis keloid 51  
 furunculosis 249

- Bacterial infections of scalp**—Cont d  
 impetigo contagiosa 245  
 of Bockhart 245  
 infectious eczematoid dermatitis 266  
 leprosy 270  
 lupoid syphilis 253  
 malignant pustule 265  
 perfolliculitis capitis abscedens et sufficiens 260  
 superficial pustular perifolliculitis 245  
 tuberculous 267
- Bactericidal and antiseptic preparations**  
 formulary 550
- BAL** 135
- Baldness (see also Alopecia)**  
 classification of scalps 174 178  
 common 173  
 types of sketches 175
- Barbiturates** as cause of dermatitis 421
- Basal cell epithelioma of scalp** 511  
 clinical appearance 517  
 histology 512  
 treatment 513 514

- Basal squamous cell epithelioma** 516
- Bayonet hair** 39
- Bearded hair** 39
- Benign connective tissue new growths** 497  
 cystic epithelioma multiple 488  
 epithelial new growths 478
- Bismuth therapy** in lupus erythematosus 466
- Black point** attached to atrophic hair 361
- Blastomycosis** 315 346
- Bleaches** hair 144
- Blue nevus** 507 503
- Bockhart impetigo** of 215
- Bombs** atomic effect on scalp 192
- Brilliantines** 141  
 formulary 554
- Brush types** of for brushing hair 172
- Bromides** as cause of dermatitis 41
- Brushing of hair** 177
- Burns** as cause of central alopecia 188
- Burrows** solution in treatment of furuncle or carbuncle 251  
 of impetigo of Bockhart 248  
 of lupoid syphilis 55

## C

- Calcifying epitheliomas of scalp** 518
- Calcosis** 457
- Calgon** for hair water 126
- Canties** 74 75 76
- Carbohydrates** effect on scalp 109
- Carbon dioxide solid** as treatment of keloid 504  
 in therapy of angioma 501
- Carbuncle** typical 251
- Carbuncles** of scalp surgical considerations 477
- Carbunculous** clinical features 219  
 etiology 251  
 pathology 249  
 therapy 251
- Carcinoma adnexal histology** 519  
 dermal appendage 517  
 in oiling between scalp areas 189





## Diseases—Cont d

- systemic, scalp involvement due to (*see* Systemic diseases involving scalp)
- Dissecting cellulitis of scalp, clinical features, 260
  - differential diagnosis, 263
  - etiology and pathology, 264
  - therapy, 264
- Disseminated cutaneous herpes simplex, 285
- neurodermatitis, 414
- Distribution of hair color, 67
  - forms, 66
  - regional, and arrangement patterns of hair, 61
- Dressings for surgery of scalp, 478
- Dry shampoos, 127
- Dyes, hair, 143
  - medicolegal aspects, 196
  - metallic, 145
  - synthetic organic, 145
  - vegetable, 144
- Dysplasia, ectodermal, hereditary, 166

## E

- Eccrine sweat glands, anatomy of, 56
- Ectodermal defect, congenital, 168
  - hypotrichotic, 163
  - dysplasia, hereditary, 166
- Eczema, atopic, 414
  - infantile, 236, 238
  - treated by dietary measures, 239
- seborrheic dermatitis differentiated from, 236
- Eczematoid dermatitis, infectious, 266
- Eczematous pyodermititis, 266
- Egg yolk shampoos, 128
- Elasticity of hair, study of, 84, 92
- Electrodesiccation for folliculitis keloidalis, 260
- Electrosurgery of angioma, 501
- Embryology of hair growth, 21
  - of human being, 28
  - of rat and mouse, 21
- Emotional disturbances as cause of alopecia areata, 369
- En coup de sabre, lesion of scleroderma, 469, 470, 471
- Endocrine factors as cause of alopecia areata, 372, 373
- Endocrines and hair growth, 96
  - in etiology of male pattern alopecia, 181
- Epidemic exfoliative dermatitis, 423
- Epidermoids, 479
- Epidermolysis bullosa, 421, 425
- Epilation, x ray device, 328
- Epithelial cysts, 479
  - nevi, 482
  - new growths, benign, 478
  - malignant, 510
- Epithelioma adenoides cysticum, 488
  - basal cell, 511
  - basal squamous cell, 516
  - calcifyine, 518
  - multiple benign cystic, 488
  - squamous cell, 527
  - transitional type histology, 520
- Epitheliomatosis, superficial, 529

## Erysipelas, 266

- Erythema annulare centrifugum (erythema multiforme), 425
  - multiforme, 425, 426
  - of scalp margins, 425
  - nodosum, 426
- Erythredema as cause of trichotillomania, 423
- Erythroderma, 426
  - desquamativa, 426
  - ichthyosiform, 428
  - tumors, and alopecia in mycosis fungoides, 534
- Estrogens and hair growth, III
  - in limitation of dandruff, 235
- Exanthems, 426
- Exclamation point hairs, 360, 362
- Exfoliative dermatitis, 426
  - caused by barbiturates, 421
  - epidemic, 423
- Extensometer, 84, 87

## F

- Facial hair, 71
  - hirsutism and adrenogenital syndrome, 101
- Fats, effect on scalp, 109
- Favus, clinical features, 332
  - differentiated from pseudopelade, 204
  - etiology, 337
  - lupus erythematosus differentiated from, 461
  - pathology, 336
  - therapy, 340
- Female pattern alopecia, 178
- Fetal hair, distribution of, 63
  - steps in development of, 29, 30
- Fibromas of scalp, 505
- Fibrosarcoma of skin, 530
- Fistula, auricular, congenital, 171, 172
- Fixatives and hair creams, 141
  - nonoily, formulary, 553
- Flocculation tests, cephalin cholesterol, results in, 140
- Follicle, hair, anatomy of, 34
- Follicular atrophoderma, 169, 170
  - lichen planus (lichen pilaris seu spinulosus), 432
  - plugging of scalp, 433
- Folliculitis de la forme aigue miliaire, 247
  - decalsans, 208, 209, 210, 211
  - clinical features, 208
  - differentiated from pseudopelade, 204
  - etiology, 213
  - lupus erythematosus differentiated from, 461
  - therapy, 213, 216
  - keloidalis clinical features, 256
  - etiology and pathology, 259
  - therapy, 259
- Forelock, white, 80
- Formalin in scalp preparations, 171
- Formulary, 542
- Frambesia, 284
- Frambesiform syphilide of scalp, 276
- Fungicides formulary, 551
- Fungal infections, favus, 332
  - of hair and skin etiology, 313
  - pathology, 309
  - of scalp, 287

## Fungus infections—Cont d

- pedra 341
- systemic 346
- therapy 322
- trichomycosis 287
- Furunculosis clinical features 249
- etiology 251
- pathology 249
- therapy 251

## G

- Genetic factors in common baldness 181 182
- Giant follicular lymphoblastoma 533
- Glands of internal secretion and hair growth 96
- sebaceous anatomy of 49
- sweat anatomy of 55

(  
(  
(  
I

- therapy 305
- Granulomatous congenital auricular fistula 172
- Grayning of hair 75 79
- localized following herpes zoster of supraorbital nerve 80
- premature 80

## H

- Hair and scalp influence of nutrition on 109
- animal types of 194 195
- arrangement and distribution 61
- bayonet 39
- beaded 39
- bleaches 144
- brushing of 122
- color distribution of 67
- combing of 123
- creams and fixatives 141
- nononly formulary 553
- cuticular scales and medulla of 67
- cutting of 124
- dressings formulary 552
- dyes 143
- dermatitis following use of 150
- medicolegal aspects 196
- metabolic 145
- synthetic organic 145
- vegetable 144
- exchange and growth 94
- facial 71
- features medicolegal 197
- fetal distribution of 63
- follicle anatomy of 34
- follicle on of 57
- forms distribution of 66
- gross 65
- frequency of washing 15
- growth ACTH and 105
- adrenal cortex and 104

## Hair growth—Cont d

- and development three factors in 95
- and hair exchange 94
- corrosive and 105
- effect of menopause on 102
- of pregnancy on 101 102
- embryology of 21 28
- fetal 29 30
- endocrines and 96
- gonads and 98
- hormones and 97
- parathyroid gland and 109
- pituitary gland and 106
- thyroid gland and 108
- index 70
- knocking of acquired progressive 47 49
- knottling of 41 43
- loss rate of 178
- lost on dermatitis following use of 152
- lotions 129 130
- formulary 544
- ne
- i
- pigmentation of 72
- preparations 126
- rheologic properties 83
- ringed 81 82 83
- rinses 143
- shaft anatomy of 35
- deviations from normal 39
- variation of size of 62
- shampooing 125
- shape of 70
- straightening of 124
- straighteners 142 152
- dermatitis following use of 153
- twisted 43 46 47
- types of study of 193 200
- variation of 72
- weight 70
- Hallopeau acrodermatitis continua 412
- trichotillomania 387
- Hand-Schüller-Christian disease 457
- Harlequin fetus showing element of scalp 427
- Hatband dermatitis 154
- Heat excessive as factor in cicatricial alopecia 188
- Helical hair 67
- Hemangiomatous 499
- Henna as hair dye 144
- Hereditary anhidrotic ectodermal dysplasia 166
- Heredity as factor in alopecia 9
- in male pattern alopecia 181
- Herpes zoster and simplex 286
- of supraorbital nerve localized ganglionic of hair following 80
- Hidrotic ectodermal dysplasia 166
- Hirsutism facial and adrenogenital syndrome 103
- Hodgkin's disease 531
- Hollander treatment of lupus erythematosus 466
- Hormones and hair growth 97
- in treatment of baldness 183

Horn cutaneous 478 479  
 Hydrotic ale with p rphyr = 467 468  
 Hyperhidrosis capitis 56  
 Hypotrichotic congenital ectodermal defect 163

## I

Ichthyosis erythroderma 478  
 Ichthyosis 477  
   follicular 479  
 Ideal shampoo 175  
 Idiopathic hemorrhagic sarcoma ultiple 530  
 Impetigo dermatitis 266  
   with pediculous capitis 347  
 Impetigo contagiosa 245  
   neonatorum 746  
   of Bockhart clinical feature 245  
   etiology 248  
   pathology 247  
   therapy 248  
 Incisions and lacerations of scalp surgical conderations 477  
 Incontinent apigment 171 479  
 Index of hair 70  
 Indigofera n hair dye 144  
 Industrial dermatitis enenata therapy 157  
 Infections fungous systemic on scalp 346  
   of scalp 45  
   anthrax 265  
   bacterial (see Bacterial infections of scalp)  
   carbunculo 49  
   dissecting cellulitis 260  
   demonstrated cutaneous herpes simplex 285  
   erysipelas 266  
   faus 332  
   folliculitis keloid 56  
   furunculo 49  
   herpes zoster and simplex 286  
   impetigo contagiosa 45  
   of Bockhart 245  
   infestous zymotic dermatitis 6  
   lepos 70  
   loas 349  
   lipodermatosis 53  
   onion 348  
   parasitic Parasitosis and infestations  
   pediculous 346  
   pedra 341  
   scabies 348  
   sporadic (see Sporadic infection of scalp)  
   surgical onderation 477  
   syphilis 27  
   tinea capitis 287  
   tuberculo 267  
   virulent and arala 286  
   virus (see Virus infections of scalp)  
   wax 84  
 Infectious ectodermal dermatitis 60  
 Inheritance factor alopecia areata 368  
   in male pattern alopecia 181  
   of hair types 65

Innervation of scalp 59 60  
 Intracutaneous nevus 483  
 Intraepidermal nevus 487 483  
 Iodides cause of dermatitis 421

## J

Jacquet agenesia plaris 166  
 Jadassohn Tche blue nevus of 507  
 Junctional or marginal type nevus (melanoma) 483  
 Juniper turpentine scalp preparations 131

## K

Kaposi sarcoma of 530  
   varicelliform eruption of 285  
 Keloid of scalp 503  
   histology 504 505  
   therapy 504  
 Keratin polypeptide chains III 89  
 Keratoma 479  
 Keratosis 483  
   arsenical 483  
   seborrheic 484  
   senile 485  
   therapy 486  
 Keratosis blennorrhagica 428 479  
   follicular 415  
   plaris 429  
   therapy 430  
 Kerion of scalp 288 290 320  
 Knocking of hair acquired progress 47 49  
 Knitting of hair spontaneous 41 43  
 Kwell treatment of pediculous capitis 348

## L

Lacerations and incisions of scalp surgical conderations 477  
 Lacquers and lotions setting 131  
 Lanugo hair with sebaceous gland 50  
 Lawsonia inermis n hair dye 144  
 Lerner erythroderma desquamata 426  
 Leopard man 75  
 Leprosy classification 270  
   clinical features 270  
   etiology 272  
   pathology 271  
   therapy 272  
 Lesions of scalp satellite or berrant 450  
 Leucotrichia annular 81  
 Leukemia 531  
   utis 537  
 Leukodermia 531  
 Leichen chronic simplex (see Neurodermatitis circumscripta)  
   planus 431  
   follicular (lichen plaris seu spinulo) 437  
   clinical features 437  
   etiology 436  
   pathology 435  
   therapy 436  
 Lichen planus like eruption of follicles and scalp following quinine ingestion 421  
 Lipodermatosis 457

- Lipoma of scalp, 509  
   removal of, 478  
   therapy, 510  
 Lipomelanotic reticulosis, 436  
 Liquid shampoos, 127  
 Local anesthesia for surgery of scalp, 478  
 Localized sarcoma, 530  
 Loiasis, 319  
 Lotions and lacquers, setting, 131  
   hair, 129, 130  
   formulary, 544  
   scalp, formulary, 543  
   wave, 132  
 Lupoid syphilis, clinical features, 253  
   etiology and pathology, 255  
   therapy, 255  
 Lupus erythematosus differentiated from  
   pseudopelade, 204  
   from seborrheic dermatitis, 236  
 Lupus erythematosus (discoid type), clinical  
   features, 458  
   differential diagnosis, 461  
   etiology, 465  
   histology, 462, 463, 464  
   pathology, 461  
   therapy, 465  
 Lupus erythematosus disseminatus, 467  
   vulgaris, 268  
 Lymphadenosis cutis circumscripta, 532  
   universalis, 533  
 Lymphangiomas of scalp, 502  
 Lymphatic circulation of scalp, 58, 59  
 Lymphoblastoma, 531  
   cutis, 78  
   giant follicular, 533
- M**
- Male pattern alopecia (*see* Alopecia, male  
   pattern)  
 Malignant connective tissue neoplasms, 529  
   epithelial new growths, 510  
   pustule, 265  
 Mapharsen in treatment of lupus erythema-  
   tosis, 466  
 Marginal alopecia, traumatic, 185, 186, 187  
   type nevus (melanoma), 483
- N**
- Naphuride in treatment of lupus erythema-  
   tosis, 466  
 Negro, baldness in, 72  
   facial hairs of, 71  
   kinky hair of, 65, 66  
 Neoplasms, 476 (*see also* New growths)  
   connective tissue, malignant, 529  
 Nerves of scalp, 59, 60  
 Neurodermatitis, circumscribed, clinical fea-  
   tures, 375  
   differential diagnosis, 379  
   etiology, 382  
   pathology, 380  
   therapy, 383  
   disseminated, 414  
 Neurodermatoses (*see* Psychogenic scalp dis-  
   orders)  
 Neurofibroma of scalp, 505  
   histology, 507  
   therapy, 507  
 Neurofibromatosis of von Recklinghausen, 506  
 Nevocarcinoma (melanotic or amelanotic),  
   522  
   histogenesis, 523  
   treatment, surgical, 526  
 Nevus araneus, 502  
   blue, 502, 503  
   cutis type (intracutaneous), 483  
   epithelial, 482  
   therapy, 483  
   epitheliomatocylindromatosis, 489, 490,  
     491, 492  
   therapy, 493  
   epitheliomatosis sebaceous capitis, 491  
   flammeus, 497  
   intraepidermal, 482, 483  
   junction or marginal type (melanoma), 483  
   pigmented, 81  
   syringadenomatosis papillifera, 491  
   vascular, 497  
   woolly hair, 496  
 New growths, 476  
   connective tissue, benign, 497  
   angioma, 497  
   blue nevus, 502  
   cicatrx and keloid, 503  
   fibromas, 505  
   cranuloma pyodermicum, 507

New growths, connective tissue, benign—  
Cont'd

- lipoma, 509
- myxoma, 510
- osteoma, 510
- scars, 503
- vascular nevi, 497
- malignant, 529
  - fibrosarcoma, 530
  - Hodgkin's disease, 531
  - Kaposi's sarcoma, 530
  - leukemia, 531
  - lymphoblastoma, 531, 533
  - mycosis fungoides, 534
  - sarcoma cutis, 529
- epithelial, benign, 478
  - cutaneous horn, 478
  - cysts, 479
  - keratoses, 483
  - molluscum contagiosum, 486
  - multiple benign cystic epithelioma, 488
  - nevi, 482, 489, 494, 496
  - psammoma, 495
  - syringoma, 495
  - verruca, 496
- malignant, 510
  - basal cell — 511

## surg:

burgery of scalp)

Nononly fixatives and hair creams, formulary, 533

Normal scalp, care of, 122

Nutrition, influence on hair and scalp, 109

## O

Oils, hair, formulary, 553

Ointments, formulary, 548

131

## P

Pachyonychia congenita 172

Panthenol therapy, 80

Para aminobenzoic acid gray hair and 77  
78, 79

in treatment of scleroderma 472

Paraphenylenediamine as hair dye 146

Parapsoriasis, 436

Parasites, animal, 346

as cause of alopecia areata, 371

vegetable (fungal), 287

Parasitic infections and infestations 287

favus, 332

loiasis, 349

onchocerciasis, 348

pedra, 341

scabies, 348

tinea capitis, 287

Parathyroid gland and hair growth, 108

Patch tests, aim of, 154

controls, 155

exposure, 156

interpretation, 156

material, 155

medicolegal aspects, 157

method, 154

reactions, negative, 157

positive, 156

site, 155

strength of test material, 155

to show sensitivity to cold wave prepara-  
tions, 135-140

usage studies, 157

Pediculosis capitis, 346

alopecia and pyoderma secondary to, 347

local remedies, 348

Pediculus capitis, 346

Pemphigus of scalp, 436, 437

vegetans, 438

Percutaneous absorption and permeability, 92

Perifolliculitis capitis abscedens et suffodiens

(see Dissecting cellulitis of scalp)  
superficial pustular (see Impetigo of Bock-  
hart)

Permanent waving, 132, 133

cold wave preparations, 134

Permeability of skin and scalp, 92

Pernicious anemia, 467

Philips tube (contact therapy) in angioma  
500

Physical agents, cicatricial alopecia due to,  
184

Physiology of hair, 72

Pickard albinism, 75

Piedra, clinical features, 341

etiology, 342

pathology, 342

therapy, 344

Piedraia hortai, 342, 343

Pigmentary dermatosis, incontinentia pig-  
menti, 429

sypilide of secondary syphilis, 275

Pigmentation of hair, 72

ringed hair, 81, 82, 83

Pigmented nevus, 81

intraepidermal, 482

true, 483

Pilary system, development of, 21

Pili annulati, 81

torti, 43, 46

Pilometer, 84

diagram of, 85

in operation, 86

Pilometric measurements, 71

393

circinata, 233

rosea, 438

seborrheic dermatitis differentiated from,  
239

rubra pilaris, 439

simplex and steatoides, 220

clinical features, 220

- Pityriasis simplex and steatoides—Cont'd**  
 etiology, 224  
 pathology, 222  
 therapy, 225  
 steatoides as cause of seborrheic dermatitis, 240
- Pityrosporum ovale**, 222, 223, 239
- Poliosis**, 74, 81
- Polypeptide chains of keratin**, 88, 89
- Porphyria**, 467
- Port wine stain (nevus flammeus)**, 497
- Powdered shampoos**, 126
- Pregnancy**, effect on hair growth, 101, 102
- Preparations**, cold wave, 134  
 for use on hair and scalp, 126
- Pressure dressings for surgery of scalp**, 478
- Propylthiouracil as cause of alopecia**, 421
- Proteinosis**, lipid, 457
- Proteins**, effects on scalp and hair, 110
- Pruritus**, clinical features, 396  
 diagnosis, 397  
 etiology, 398  
 ointments and lotions, formulary, 551  
 pathology, 397  
 preparations for local treatment, 403  
 therapy, 401
- Psammoma**, 495
- Pseudopelade**, clinical features, 200  
 etiology, 205  
 lupus erythematosus differentiated from, 461  
 pathology, 204  
 sclerosis of follicle, 206  
 therapy, 205, 208  
 with chondrodystrophia calcificans congenita, 169
- Psychosis**, 254  
 and  
 from,  
 of scalp, preparations, formulary, 552  
 pathology, 445  
 seborrheic dermatitis differentiated from, 235  
 therapy, 446
- Psychoanalysis**, only therapy for acarophobia, 393
- Psychogenic scalp disorders**, 356  
 acarophobia, 392  
 alopecia areata, 357  
 circumscribed neurodermatitis, 375  
 lichen chronicus simplex, 375  
 pityriasis amiantacea, 393  
 pruritus, 396  
 suboccipital dermatitis, 375  
 tinea amiantacea, 393  
 trichotillomania, 386
- Psychosomatic aspects of scalp disease**, 356
- Psychotherapeutic measures of Stokes**, 384  
 385

## Q

- Quinacrine as cause of dermatitis**, 421
- Quinine hydrochloride as cause of alopecia**, 421  
 in scalp preparations, 130
- Quinoline preparations in treatment of impetigo of Bockhart**, 248
- Quinolone compound ointment**, 412  
 in treatment of furuncle and carbuncle, 252  
 of lupoid syphilis, 255
- R**
- Racial variations in hair forms**, 66
- Radium and roentgen rays as cause of alopecia**, 189  
 rays in treatment of angioma, 501  
 therapy of keloid, 504
- Rain water for shampooing hair**, 126
- Rat and mouse**, development of pilary system in, 21
- Reactions to patch tests**, evaluation of, 156, 157
- Resorcinol in scalp preparations**, 131
- Rheologic properties of hair**, 83
- Ringed hair**, 81, 82, 83
- Ringworm**, "gray patch," 288
- Rinses**, hair, 143  
 shampoo, 129
- Rodinal**, 146
- Roentgen and radium rays as cause of alopecia**, 189
- Rosacea like tuberculid**, 270
- Rubella**, lesions on scalp caused by, 426

## S

- Salicylic acid in scalp preparations**, 130
- Sarcoidosis**, 268
- Sarcoma cutis**, 529  
 localized, 530  
 multiple idiopathic hemorrhagic, 530
- Satellite lesions of scalp with other diseases**, 450
- Savill**, epidemic exfoliative dermatitis, 423
- Scabies**, 348
- Scalp**, anatomy of, 31  
 and hair, influence of nutrition on, 109  
 arterial supply of, 57  
 chronic scaly disorders of, formulary, 518, 519  
 defects of, congenital, 168  
 dermatitis of, 148  
 therapy, 157  
 disorders of psychogenic origin (see Psychogenic scalp disorders)  
 effect of vitamin deficiencies on, 111  
 folding of, complication of dermatitis, 158  
 hairiness, sketches of categories of, 175  
 infections (see Infections of scalp)  
 innervation of, 59, 60  
 lotions formulary, 513  
 lymphatic circulation of, 58, 59  
 musculature of, 60  
 normal, care of, 122  
 preparations, 130

## Scalp—Cont'd

- skin diseases involving (*see* Skin diseases involving scalp)
- sweating of, excessive, 56
- systemic diseases involving (*see* Systemic diseases involving scalp)
- types of, in classification of baldness, 174-178
- venous drainage of, 58
- Scaly disorders of scalp, lotions, formulary, 548
- ointments, formulary, 549
- Scarlet fever, lesions on scalp caused by, 426
- Scars of scalp, 503
- Scleroderma (circumscribed type), clinical features, 467
- en coup de sabre, 469, 470, 471
- etiology, 470
- pathology, 470
- therapy, 471
- Scrofuloderma, 268, 269
- Scutula, typical, 332, 334, 335
- Sebaceous cysts, 480, 481
- of scalp, removal of, 477
- glands, anatomy of, 49
- normal, 51
- Seborrhea in etiology of male pattern alopecia, 182
- oleosa, 226
- as cause of seborrheic dermatitis, 240
- clinical features, 226
- etiology, 229
- lotions, formulary, 550
- male pattern alopecia with, 227
- ointments, formulary, 549
- pathology, 228
- therapy, 231
- Seborrheic bacillus, morphology of, 428
- dermatitis, 232
- cuticular alopecia due to, 230
- circatizing, 241
- clinical features, 232
- differential diagnosis, 235
- etiology, 240
- lotions, formulary, 550
- ointments, formulary, 549
- pathology, 239
- psoriasisiform type, 234
- therapy, 242
- diathesis, 220
- psoriasis simplex and seborrheic, 220
- seborrheic oleosa, 226
- seborrheic dermatitis, 232
- keratosis, 484-485
- Seborrhiasis, 235
- Sebum, constituents of, 54
- on forehead, 53
- Selenium disulfide in treatment of seborrheic diathesis, 226
- Senile keratoses, 485
- Sensory nerves of scalp, distribution of, 60
- Setting lotions and lacquers, 131
- Shaft hair, anatomy of, 45
- deviations from norm., 39
- variation in size of, 62
- Shampoo, ideal, 125
- rinses, 129
- Shampooing of hair, 125
- Shampoos, formulary, 542
- types of, 126-129
- Sickle cell anemia, 472, 473
- Simmonds' disease and hair growth, 107
- Singeing of hair, 124
- Sjogren's syndrome, 473
- Skin diseases involving scalp, 407
- acne necrotica miliaris, 407
- urticata, 408
- varioliformis, 409
- acrodermatitis continua, 412
- alopecia (nonspecific), 412
- atopic dermatitis, 414
- Darier's disease, 415
- dermatitis herpetiformis, 419
- medicamentosa, 420
- vegetans, 423
- disseminated neurodermatitis, 414
- epidemic exfoliative dermatitis, 423
- epidermolysis bullosa, 424
- erythema multiforme, 426
- nodosum, 426
- erythroderma, 423
- erythroderma, 426
- desquamativa, 426
- exanthema, 426
- follicular lichen planus, 432
- ichthyosis, 427
- incontinentia pigmenti, 429
- keratosis blennorrhagica, 429
- pilaris, 429
- lichen pilaris seu spinulosus, 432
- planus, 431
- lipomelanotic reticulosis, 436
- parapsoriasis, 436
- pemphigus, 436
- pityriasis rosea, 438
- rubra pilaris, 439
- psoriasis, 440
- satellite or aberrant lesions, 450
- urticaria, 450
- pigmentosa, 452
- xeroderma pigmentosum, 452
- manifestations, systemic diseases with, 457
- Skull depressions, congenital, 169
- Soap shampoos, 126-127
- Soapless shampoos, 128
- Solutions, permanent wave, 133
- Spider nevus, 502
- Spiral hair, 66
- Spirochetal infections of scalp, 272
- frambesia, 284
- sypilis, 272
- yaws, 284
- Squalene, 54
- Squamous cell carcinoma with sebaceous structures, 517, 518
- epithelioma, 527
- histology, 528
- therapy, 528
- Staining of hair cells, 199, 200
- Staphylococcus albus, 223, 225, 230, 239, 240
- aureus, 408
- Straighteners, hair, 142, 152
- dermatitis following use of, 153
- Strawberry mark, 497

- Streptococcal dermatitis, chronic, 266  
infection of scalp differentiated from seborrheic dermatitis, 236
- Stress as cause of alopecia areata, 371
- Suboccipital dermatitis (*see* Neurodermatitis, circumscribed)
- Sulfone therapy for leprosy, 272
- Sulfur in scalp preparations, 130
- Superficial carcinoma (superficial epithelioma), 329  
pustular perifolliculitis (*see* Impetigo of Bockhart)
- Surgery of scalp, 476  
anesthesia, local, 478  
dressings, 478  
general considerations, 476  
infections, 477  
lacerations and incisions, 477  
removal of tumors and cysts, 477
- Sweat glands, anatomy of, 55
- Sweating of scalp, excessive, 56
- Sycosis, lupoid, 253
- Syndrome, Werner's, 172
- Synthetic organic dyes for hair, 145
- Syphilide, frambesiform, of scalp, 276  
pigmentary, 275  
serpiginous, ulcerative, 278
- Syphilis, alopecia of, 273, 274, 275  
clinical features, 272  
etiology, 284  
pathology, 283  
tertiary, 277  
therapy, 284
- Syphilitic destruction of face with involvement of scalp, 279
- Syringoma (*syringocystadenoma*), 495
- Systemic diseases involving scalp, 457  
calcinosis, 457  
Hand Schüller Christian disease, 457  
lipoid proteinosis, 457  
lupus erythematosus (discoid type), 458  
disseminatus, 467  
pernicious anemia, 467  
porphyria, 467  
scleroderma (circumscribed type), 467  
sickle cell anemia, 472  
Sjogren's syndrome, 473  
Vogt-Koyanagi syndrome, 473  
xanthoma tuberosum, 474  
fungus infections, 346

## T

- Telangiectasias of scalp, 502
- Tensile strength of hair, study of, 84
- Tension, scalp, as cause of alopecia, 179, 180
- Tertiary syphilis of scalp, gumma of, 277, 278
- Tests, patch, in diagnosis of dermatitis, 154  
to show sensitivity to cold wave preparations, 136, 137, 138, 139, 140
- Thallium salts as cause of alopecia, 421, 422, 423
- Therapy of dermatitis venenata, 157  
of male pattern alopecia, 183
- Thioethylenes in cold wave preparations, 133, 135

- Thorium X in treatment of alopecia areata, 374  
of circumscribed neurodermatitis, 383  
of port wine stain, 497
- Thyroid gland and hair growth, 108
- Thyrotoxicosis as cause of alopecia areata, 373
- "Tic d'epilation," 387
- Tinea amiantacea (*see* Pityriasis amiantacea) capitis, clinical features, 288  
differential diagnosis, 290  
public health aspects of, 331  
therapy, 322  
x ray, 324  
types of, 288
- Toxicity as cause of alopecia areata, 372
- Transitional type of epithelioma, histology, 520

- Trichoclasia, 42
- Trichoclasmania, 387
- Trichoeptithelioma, 488
- Trichokryptomania, 387
- Trichonodosis, 41, 43
- Trichophyton acuminatum, 301, 305, 306, 312  
cerebriforme, 304, 318  
crateriforme, 291, 301, 307  
faviforme, 309, 314, 316, 317  
gypseum, 294, 308, 312  
purpureum, 294, 319  
sulfureum, 304, 308  
type of tinea capitis, 288  
violaceum, 294, 301, 304, 310
- Trichoptilosis, 43, 44, 45
- Trichorrhexis nodosa, 43, 44, 45  
differentiated from tinea capitis, 291
- Trichosporum beigeli, 342, 343  
giganteum, 342, 344
- Trichostasis spinulosa, 44, 48
- Trichotillomania and trichokryptomania differentiated from tinea capitis, 290  
clinical features, 386  
differential diagnosis, 387  
erythroderma as cause of, 423  
etiology, 387  
extensive, 390  
in psychotic individual, 391  
therapy, 387  
with excoriations of scalp, 388
- Tuberculosis colliquativa, 268, 269  
cutis papulonecrotica, 268, 269  
verrucosa, 268  
miliaris disseminata faciei, 270  
of scalp, 267
- Tumors, 476 (*see also* New growths)  
and cysts of scalp, removal of, 477
- Turban tumor, 489, 490, 491, 492  
therapy, 493
- Twisted hairs (pili torti), 43, 46
- Types of scalp, baldness and, 174, 178

## U

- Ulerythema ophryogenes, 430  
syconiforme, 254



Ultraviolet light causing burns of scalp, 189  
in treatment of alopecia areata, 374  
of psoriasis of scalp, 448  
of seborrhea oleosa, 232  
of tinea capitis, 291  
Urticaria of scalp, 450  
pigmentosa, 451, 452

## v

Variation of hairs on head, 72  
Varicella and variola, 286  
    lesions on scalp caused by, 426  
Variola, lesions on scalp caused by, 426  
Vascular nevus, 497  
Vegetable dyes, hair, 144  
    parasites, fungi, 287  
Vellus, 61, 62  
Venous drainage of scalp, 58  
Verruca (wart), 496  
    senilis, 484  
Virus infections of scalp, 285  
    disseminated cutaneous herpes simplex,  
        285  
    herpes zoster and simplex, 286  
    Kaposi's varicelliform eruption, 285  
    varicella and variola, 286  
Vitamin A and scalp, 111, 114  
    in control of pityriasis simplex and  
        steatoides, 226  
    in therapy of pityriasis rubra pilaris, 440  
B and scalp, 115  
Bu in therapy of seborrheic dermatitis, 242

466

## W

- Warts on scalp, 496
- Washing of hair, 125
- Water for shampooing of hair, 125
  - softening, 126
- Wave lotions, 132
  - preparations, cold, 134
  - reactions to, 135, 136, 137, 138, 139, 140
- Waving lotions, effects of, 139
  - permanent, 132, 133
- Weight of hair, 70
- Werner's syndrome, 172
- White forelock, 80
- Witkop, 282, 283
- Wood's light in treatment of tinea capitis, 291
- Woolly hair nevus, 496

x

- Xanthoma tuberosum, 474
- Xeroderma pigmentosum, 452, 529
  - with carcinomatosis, 451
- X ray therapy as cause of cicatricial alopecia, 190, 191
  - contact, of angioma, 300
  - of dermatitis of scalp, 158
  - of favus, 340
  - of folliculitis keloidalis, 260
  - of keloid, 504
  - of keratoses, 486
  - of psoriasis of scalp, 448, 449
  - of seborrhea oleosa, 232
  - of squamous cell epithelioma, 528
  - of tinea capitis, 324
    - dangers, 329
    - device, 328
    - dose, 326
    - public health aspects, 331

## Y

Yawz. 284

